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MUSCLE DYSTROPHIES AND ATROPHIES OCCURRING IN CHILDHOOD: FROM THE ORTHOPÆDIC ASPECT.

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CLASSIFICATION.

IN order to obviate a long list of apparently meaningless names I have attempted to classify the conditions under consideration into two groups, according to whether the pathological changes usually manifest themselves before or after birth. When more is known of the Group II series, some of the conditions now classified under that heading may have to be transferred to Group I.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 25, 1938.

Group I:

1. Congenital high shoulder (Sprengel's shoulder).
2. Tibial kyphosis (congenital angulation of the tibia).
3. *Myodystrophia fœtalis deformans (arthrogryposis multiplex congenita).*
4. Congenital torticollis.
5. *Amyotonia congenita.*

Group II:

1. Pseudo-hypertrophic muscular paralysis (Duchenne type).
2. The juvenile type of Erb.
3. Facio-scapulo-humeral type of Landouzy and Déjerine (sometimes present at birth).
4. The distal type.
5. Peroneal muscular atrophy.
6. Anterior poliomyelitis.
7. Volkmann's contracture.

GROUP I.

Congenital High Shoulder.

Middleton has thrown much light on most of the conditions listed in Group I. He finds that con-

genital high shoulder results from the failure of a mass of muscle connecting skull to scapula, as well as all the muscles of the shoulder girdle, to develop beyond the myoblastic stage, and the presence of excessive fibrous tissue in those muscles, which fibrous tissue not infrequently ossifies. The scapula is thus anchored in its primitive position and fails to descend as the fœtus develops. It is interesting to note also that the lower part of the trapezius muscle is absent in these cases. To quote one sentence from Middleton's paper.

> In congenital high shoulder we are dealing with a widespread lesion of mesodermal development in the cervical region of the embryo; this shows itself in irregular vertebral segmentation and in muscular abiotrophy in the affected area of the limb bud.

In view of these findings, it is not surprising that attempts to correct the deformity surgically are usually disappointing.

Tibial Kyphosis.

The same writer considers that tibial kyphosis, with which is associated a persistent *pes equinus*, is primarily due to the failure of the calf muscles to grow in length, *in utero*, even though fully differentiated, the deformity of the tibia being produced before that structure is ossified.

No change was noted in the histological structure of the calf muscles. Absence of the fibula, congenital dislocation of the hip, and other deformities may accompany this abnormality. Treatment consists in elongation of the *tendo Achillis* and an osteoclasis or osteotomy of the tibia and splinting with the foot at right angles and the knee straight.

Myodystrophia Fœtalis Deformans.

Myodystrophia fœtalis deformans affects upper or lower limbs or both upper and lower limbs, and is accompanied by any or all of the following disabilities: (i) congenital *genu recurvatum*, (ii) club foot or club hand, (iii) congenital dislocation of the hip, (iv) absence of the patella, (v) limitation of joint movements, (vi) flexion deformity of joints, especially of the hips and knees. The muscles are much wasted, and cannot be expected to recover.

Pathology.

The muscles examined by Middleton appeared as a thin belly of fibro-fatty tissue, with no appearance of normal muscle, whilst microscopically they were seen to be composed of fat and some fibrous strands, with very little muscle tissue. Middleton considered the condition to be due to a process of degeneration of formed and differentiated muscle. As a rule the limbs only are affected.

Treatment.

The club foot and club hands associated with this condition are particularly resistant to treatment and readily relapse. Nevertheless, one must persevere with manipulations and splinting for many years in order to maintain as good a position as possible, so that any bone operation at the age of fourteen years will be made easier and more effective.

Middleton suggests that the hip dislocations are due to traction by the fibro-fatty gluteal muscles as the pelvis is growing in size. Such dislocations are impossible to reduce without an extensive muscle-stripping procedure. It is probably wiser to leave them unreduced, and when the patient has reached the age of eighteen or so, to perform a simple osteotomy to correct the stance.

The reduction of the *genu recurvatum* may be accomplished by a series of manipulations, followed by adequate splinting; whilst in gross cases open elongation of the quadriceps tendon will be necessary. The flexed knees and hips should be slowly straightened on Thomas's bed knee splints and the Jones's frame, respectively.

It has been noted that fœtal movements are absent in these cases and parturition has been rendered difficult by the deformity and rigidity of the limbs.

Congenital Torticollis.

The theories concerning the ætiology of congenital torticollis are legion; the one in favour at present, and which seems to have reasonably sound support, is that it is due to a venous obstruction before birth. This theory places it in the same category as Volkmann's contracture.

The venous obstruction leads to a round-cell infiltration, which is followed by the deposition of fibrous tissue, with destruction of muscle fibres. The deformity then develops as the fibrous tissue contracts.

Treatment.

If the patient is seen soon after birth, that is, in the sternomastoid tumour stage, repeated stretching of the affected muscle should be carried out for a prolonged period. If the patient is seen after fibrosis has taken place, open division of both heads of the sternomastoid muscle and the deep cervical fascia is required. This must be followed by daily stretching and active exercises.

Amyotonia Congenita.

The two features of *amyotonia congenita* are (i) the generalized muscle weakness, (ii) the tendency to improve, and on these the principles of treatment are based.

Treatment.

At all times prevent overstretching of capsule and ligaments on one side and structural shortening of the components on the opposite side of the joint, by splinting, which should be continuous, except when exercises are being carried out.

When recovery has progressed sufficiently, ambulation, aided by very light walking callipers, spinal brace and a Thomas's collar, should be encouraged. Shoes should be adjusted so as to prevent stretching of the longitudinal arch of the foot and the internal lateral ligament of the ankle joint.

GROUP II.

Pseudo-Hypertrophic Muscular Paralysis.

Pseudo-hypertrophic muscular paralysis, with its allied forms, the juvenile type of Erb, the facio-scapulo-humeral type, and the distal type, may be considered together with peroneal muscular atrophy in so far as their orthopædic treatment is concerned.

At this stage let me issue a plea not to regard these cases as hopeless, even though we know that the majority of the patients suffering from most of the above-mentioned conditions do slowly retrogress. Occasionally we find that the disease remains stationary for a shorter or longer interval, and then we regret not having prevented the development of deformities. Even if we anticipate an ultimately fatal termination, it is our duty to keep such patients on their feet as long as possible. This can be done by means of night splints to retain joints in their optimum position and by means of inside or outside leg-irons, toe-straps, adjusted shoes, callipers, Thomas's brace and collar, shoulder-straps and wrist splints, fitted if necessary with a finger-extending mechanism according to the particular needs of each case. Certain patients, when the disease ceases to progress, may be benefited by muscle transplantation or even stabilizing operations on the bones about certain joints. Should the patient present himself with fixed deformities already present, for example, *pes equinus* or flexion of a knee, one must correct these deformities, conservatively if possible, and then prescribe suitable splinting to prevent recurrence as much as possible.

Anterior Poliomyelitis.

With regard to the orthopædic treatment of anterior poliomyelitis, it is sufficient to say that efficient splinting is still of prime importance in all stages of the disease, whilst muscle reeducation should be given twice daily at least.

Volkmann's Contracture.

Volkmann's contracture is now recognized to be the result of venous obstruction. If the patient is seen within 36 hours of the onset, incision through the deep fascia of the antecubital fossa and evacuation of any blood clot may avert disaster.

Once the deformity has developed, as much correction as possible should be obtained by conservative means, after which the question of stripping the origin of the flexor group of muscles from the internal epicondyle will have to be considered if further improvement is to be obtained.

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MUSCULAR ATROPHIES AND DYSTROPHIES IN CHILDHOOD.[1]

By R. J. TAYLOR,
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As the time at my disposal is limited, I propose to describe briefly the system diseases and merely mention that muscular atrophy may be caused by a great number of conditions, such as disuse, prolonged pyrexia and cachexia, various forms of peripheral neuritis, including diphtheria, infantile paralysis, birth palsies, syringomyelia, *spina bifida*, spinal caries and tumour, pink disease and Still's disease, *myositis fibrosa*, progressive hypertrophic polyneuritis *et cetera*.

Infantile Muscular Atrophy.

Infantile muscular atrophy of spinal origin may be (i) Werdnig-Hoffmann's disease (progressive spinal muscular atrophy of children) or (ii) Oppenheim's disease (*amyotonia congenita*).

Werdnig-Hoffmann Disease.

In Werdnig-Hoffmann disease, paralysis may appear at any time during the first year, but more often appears a few weeks after birth. The proximal muscles are affected before the peripheral. There is a steady progression of the paralysis to include all the muscles of limbs and trunk and finally the bulbar muscles. Death generally occurs in a few weeks or months. The condition is familial and the sexes are equally affected.

An apparently healthy and intelligent child who has made normal progress during the first few weeks or months of life begins gradually to lose power. If the child has already stood it loses that power, then cannot sit up, and finally cannot stay sitting up if placed in that position. Weakness is first noticed in muscles of hip, then back, shoulders, thighs, arm, forearm and leg, in that order. Paralysis of proximal as compared with distal muscles is striking, as the child is able to move its fingers and toes while the arms and legs are completely paralysed.

There are no sensory disturbances. The tendon reflexes disappear. The mental condition is unimpaired. Although the muscles show paralysis of the lower motor neurone type there is not the marked hypotonia seen in *amyotonia congenita*; the arms and legs cannot be placed in grotesque positions as in the latter condition.

Oppenheim's Disease.

Oppenheim's disease (*amyotonia congenita*) has as its characteristic features extreme weakness and lack of tone of all the muscles; the arms and legs can be placed in contortionist positions and the child will lie in whatever attitude it is placed in. The weakness is usually noticed at birth. All movements can be performed, but weakly. The deep reflexes are absent or greatly diminished. There are no sensory

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 25, 1938.

disturbances and the mental condition is normal. The disease is rarely familial and shows a tendency in many cases to gradual improvement. Most patients are carried off in childhood by intercurrent infections.

The pathological histology of Werdnig-Hoffmann disease is identical with that of *amyotonia congenita*. It consists of degeneration and atrophy of anterior horn cells of the spinal cord and atrophic changes in the muscles.

The Neuritic Type of Progressive Muscular Atrophy (Peroneal Type of Charcot, Marie and Tooth).

Clinically the neuritic types resemble the myopathies; but definite pathological changes have been found in the spinal cord, namely, atrophy of the cells of the anterior horns, sclerosis of the posterior columns and slight degeneration of the pyramidal tracts. There are also degeneration and sclerosis of the nerves of the leg and forearm.

The wasting starts in the peroneal and *tibialis anticus* muscles, causing foot-drop and inversion of the feet, and slowly spreads to other muscles of the leg. The muscles below the knee are wasted while those of the thigh remain in moderately good condition. This gives the limb a bottle-shaped appearance. Later the intrinsic muscles of the hand and the muscles of the forearm may atrophy. Claw hand may develop. Sensory changes are usually present, namely, pain, hyperæsthesia, paræsthesia and loss of tactile sensation. Vasomotor disturbances are common, as also are fibrillary twitchings. The greatest difficulty is in walking; but the patient frequently overcomes this by riding a bicycle.

The disease is transmitted by males and females and both sexes may be attacked. It generally starts in childhood, but may not appear till the second or third decade. It advances slowly and usually progressively, but may halt for long periods.

The Muscular Dystrophies or Myopathies.

Under the heading of muscular dystrophies and myopathies are grouped cases in which large groups of voluntary muscles undergo primary degeneration, there being no detectable lesion in the spinal cord or peripheral nerves. Various clinical forms have been described, having regard to age of onset and groups of muscles affected; but it must be remembered that the myopathies form one great class, in many instances being hereditary and familial.

The changes in the muscles are similar in all types. Hypertrophy of the muscles may precede their atrophy, but the overgrown muscles are always weak. In many cases this hypertrophy is not a true increase of muscle tissue, but merely an increase of fat and connective tissue. Hence the term "pseudo-hypertrophy". Macroscopically the muscles appear pale red, like fish flesh. Under the microscope, in cross-section, the fibres seem to have lost their polygonal outline and appear round or oval. Some of the fibres are enlarged, others atrophied. They are generally surrounded by fibrous tissue and often separated from each other by fat. The nuclei are increased and the striation usually remains until the disease is far advanced.

I shall briefly describe the four main types of dystrophies; but it must be realized that there is no hard and fast line of distinction between them.

The Pseudo-Hypertrophic Type of Muscular Dystrophy.

The pseudo-hypertrophic type is almost entirely confined to males and is transmitted by healthy females. The age of onset is usually about four years. The boy is late in walking, is unsteady on his feet and has trouble in climbing stairs. The muscles usually hypertrophied are the deltoids, infraspinati, glutei, quadriceps, and calf muscles, while the *pectoralis major*, biceps, *latissimus dorsi* and thigh muscles are atrophied. Lordosis is a feature and the child walks with a waddling gait. He gets up in the classical way if placed on his back on the floor and when lifted by the armpits he tends to slip through the hands. The tendon reflexes persist for quite a long time, but grow weaker and disappear with the advance of the disease. The superficial reflexes remain normal. There are no sensory disturbances and the mentality is good. Fibrillary twitchings rarely, if ever, occur in the myopathies.

The Juvenile Type of Muscular Dystrophy (Scapulo-Humeral Type of Erb).

The juvenile type of dystrophy commences generally in early adolescence, but may appear at the age of six or seven years. Males and females are affected. "Winging" of the scapulæ and difficulty in raising the arms above the head are noticeable. The face and neck muscles are unaffected. The pectorales, *latissimus dorsi*, lower part of the trapezius, *serratus magnus* and rhomboids are usually atrophied. The supraspinati and infraspinati and the deltoids are unaffected or may be hypertrophied. The disease then spreads to the muscles of the arm, those of the forearm being spared. Later the muscles of the pelvic girdle and thigh become involved, while the muscles of the leg remain normal. The tendon jerks are present early but disappear as the disease progresses.

The Facio-Scapulo-Humeral Type of Muscular Dystrophy (Landouzy-Déjerine).

Weakness of the muscles of the face and shoulder girdle are characteristic of the facio-scapulo-humeral type of muscular dystrophy. In the face the *orbicularis palpebrarum* and *orbicularis oris* are chiefly affected, causing ptosis and lack of expression. Males and females are affected. In infancy there is difficulty in suckling and the child does not close its eyes when asleep. The progress of this form of the disease may become arrested for many years; but winging of the scapulæ and, later, weakness of the muscles of the pelvic girdle and thigh may occur. These children are usually able to get about much better than those suffering from the pseudo-hypertrophic type, and the disease progresses much more slowly.

The Distal Type of Muscular Dystrophy.

The distal type of muscular dystrophy is rare. The atrophy commences in the hands and feet and

slowly extends up the forearm and leg. In Gower's case the sternomastoid and facial muscles were also involved.

Myotonia Atrophica (Dystrophia Myotonica).

Myotonia atrophica is an hereditary and familial disease, occurring in both sexes and characterized by myotonia and atrophy of the facial muscles, masseters, sternomastoids, muscles of the forearm and hand and peronei. Muscular contraction persists after voluntary effort has ceased. This is especially noticed in the flexors of the fingers: the patient is unable to relax his grasp quickly. There is difficulty in chewing and walking.

Biochemical Considerations.

In recent years a vast amount of work has been done on the physiology and chemistry of primary muscular disease. The subject received a great fillip from the fortuitous discovery by Harriet Edgeworth of the efficacy of ephedrine in *myasthenia gravis*.

In an investigation of the phosphorus-holding compounds in patients with muscular dystrophy Nevin found a low total acid-soluble phosphorus content, a low creatine-phosphoric acid content of resting muscle and less than normal breakdown of this substance on stimulation. He considered this alteration as secondary to the muscle degeneration, but suggested that further investigation of the chemistry of living muscle removed at biopsy should be carried out as knowledge of the chemistry of muscle advanced.

As regards the biochemistry of the myopathies, the following conditions constantly obtain: (i) hypoglycæmia, (ii) a disturbance of the sugar tolerance, (iii) hypocholesterinæmia, (iv) creatinuria with disturbance of creatine tolerance, (v) diminution of urinary creatinine, (vi) normal blood creatine content.

As much of the study of muscular dystrophy has centred round the metabolism of creatinine, it may be as well to recall some facts with regard to creatine and muscle physiology. Nevin in a recent article has thus summarized these facts:

> Creatine is a chemical constituent of striated muscle and 98% of the creatine in the body is found in this tissue. In muscle it is linked with phosphoric acid in a highly labile compound—phosphagen. The breakdown of this substance and its resynthesis play an important part in the chemistry of muscular contraction which also involves other important reactions, namely, the breakdown of glycogen to lactic acid and the breakdown of adenosine triphosphoric acid. As a result of endogenous muscle metabolism creatinine is excreted in the urine to the extent of 23 mg. per Kilo. of body weight in 24 hours. This is the creatinine coefficient and it is a fairly constant figure for each individual, and is directly related to the total mass of voluntary muscle.
>
> In the adult male no creatine is excreted in the urine because it is all metabolized or stored in the muscles, but in children up to seven years of age and periodically in some adult women some creatine is excreted in the urine. Although the origin of the creatine in the body is not definitely known, this can only mean that in children more creatine is formed in the body than can be metabolized by the muscles at this stage in their development.
>
> In such conditions as diabetes, exophthalmic goitre and during periods of starvation and fever, creatine appears in the urine, so that increase in endogenous metabolism of muscle from different causes prevents the storage and conversion into creatinine of the normal amounts of creatine. This will also happen if a large amount of muscle tissue is lost, as through the amputation of a limb.
>
> From these observations it is to be expected that in cases of muscular dystrophy when there is considerable loss of muscle tissue with impairment of function of the remainder alteration would occur in the output of creatinine in the urine. This has been known to be decreased for a long time—since 1870.
>
> The presence of creatinuria in cases of muscular dystrophy has been known since 1909.
>
> In 1929 Brand *et al.* found that the constant creatinuria of muscular dystrophy could be increased 40% by the administration of the amino-acid glycine. This was confirmed in 1932 by Thomas *et al.* and Milhorat *et al.*, who also noted considerable clinical improvement in their patients after prolonged administration.
>
> Many conflicting reports have appeared in the literature since that time, but in no case has very definitely increased creatinuria been noted which might be regarded as an indication of improved muscular function. As the increase is only in the creatine this must mean that glycine is a precursor of creatine and leads to the increased formation of creatine in the body, which is excreted and not stored, owing to the widespread disease of the muscles, so that the administration of glycine is nothing more than an indirect creatine tolerance test.

It would seem, then, that glycine as a treatment of muscular dystrophy is based on false premises. Whatever benefit might accrue could be ascribed to overfeeding. I have never seen it do any good.

Treatment.

In the present state of our knowledge, very little can be done for muscular atrophy secondary to disease of the central nervous system. As regards primary diseases of muscles, however, a different tale can be told.

As there is a definite hereditary and familial history in the dystrophies the main attack should be along preventive lines. In the pseudo-hypertrophic type the disease occurs predominantly in males and is transmitted by apparently healthy females. The actual sufferers from the disease rarely reach sexual maturity and so do not count. Females of these families should be prohibited from marrying, while healthy males might be allowed to procreate their kind. In the families affected by other types of dystrophy, males and females alike are attacked and frequently reach sexual maturity. Marriage should therefore be proscribed to all members of these families.

In the dystrophies the lesion of the muscle fibre resembles that resulting from nerve degeneration or muscle inactivity. As direct stimulation is not an effective form of treatment, it is possible that the lesion is primarily due to absence or inadequacy of some trophic influence exerted on the muscle by the motor end-plate. This trophic influence might be the normal action of acetyl choline liberated by nerve impulses, therefore it is possible that replacement therapy might be of help. Acetyl choline itself is too rapidly destroyed in the body; but the more stable choline esters, "Doryl" and "Mecholin", might be worthy of a trial.

Eserine and its derivative, prostigmin, ephedrine and potassium ions all have identical actions in increasing the excitability of the motor end-plate. In the absence of nerve impulses they all cause

twitchings in normal subjects, the twitchings arising spontaneously in the end-plates.

All these drugs have a beneficial effect on patients with muscular dystrophies.

I have at present under treatment a girl, aged eight years, suffering from a mixed form of myopathy, with Landouzy-Déjerine features predominating, who has been taking ephedrine and potassium chloride for two years. Before treatment was instituted she could not walk fifty yards without great fatigue, could not go up or down stairs and had great difficulty in getting up when placed lying on her back on the floor. Now, apart from the characteristic final flick of the head, she gets up like a normal child, can go up and down stairs easily and can walk three miles quite comfortably. She is about to start school. I do not think this is a case of *post hoc ergo propter hoc* because improvement coincided with the beginning of the treatment, and when from any cause she has been without medicine for a couple of weeks her condition has deteriorated. I have recently given this child eserine sulphate in a dose of 0·01 gramme (one-sixth of a grain) twice a day without atropine. The mother thinks the effect of this is better still than that of ephedrine and potassium chloride. An interesting feature of this case is that at present her urinary creatine output is normal for her age and her blood sugar and blood cholesterol figures are within normal limits. There is still some diminution of urinary creatinine.

Winkelman and Moore have treated with prostigmin six patients suffering from muscular dystrophy. They noted clinical improvement in three instances, but in three advanced cases there was no response. The unpleasant side-effects noticed when prostigmin is given to adults seem not to occur in children. Therefore it is unnecessary to give atropine.

Minot, Dodd and Riven have recently treated two patients suffering from *myasthenia gravis* with guanidine (a breakdown product of creatine) and noted improvement. They consider its action comparable with that of prostigmin. It is equally efficacious whether given intravenously or by mouth, whereas the effective oral dose of prostigmin is very much greater than the parenteral. Guanidine is at present unobtainable in Australia; but when it does become available I should certainly like to try its effect in cases of muscular dystrophy.

Kolb and his co-workers have recently found that sufferers from *myotonia atrophica* could be kept free from symptoms of myotonia by the administration of quinine sulphate.

Acknowledgements.

I should like to thank Dr. J. C. Eccles, of the Kanematsu Memorial Institute, Sydney Hospital, for a very interesting dissertation on modern views of muscle physiology. I also wish to acknowledge the extensive use of Garrod, Batten and Thursfield's "Diseases of Children", third edition, 1934.

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NASAL SPRAYING WITH ZINC SULPHATE SOLUTION IN THE PROPHYLAXIS OF POLIOMYELITIS; DIPHTHERIA IMMUNIZATION AS A PARTIAL PREVENTIVE.

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IN August, 1937, when it became apparent that the outbreak of infantile paralysis in Melbourne had reached dangerous proportions, it was obvious that the disease would eventually spread to South Australia. The opportunity was thereby given to try out the technique of nasal instillation of zinc sulphate solution as described by Peet, Echols and Richter[1] and Schultz and Gebhart.[2] These investigators showed that in monkeys a 1% solution of zinc sulphate applied to the olfactory area afforded a high degree of protection against intranasal instillations of active poliomyelitis virus. Their work was done subsequent to certain other investigations in the same direction, wherein sodium aluminium sulphate, trinitrocresol, "Mercurochrome", picric acid and other substances had been used. Schultz and Gebhart particularly emphasized the necessity for adequate preparation and care in application of the method; and since in the present case ample time was available, the preliminaries of the scheme were gone into very carefully. It is to be regretted that sufficient time was not available later for the collection of more accurate records. In preparation for the campaign, which was recognized as an experiment, precautions were taken to record any difficulties that might be encountered in the application, and cards were drawn up with the following *pro forma*:

POLIOMYELITIS PROPHYLAXIS.

NAME..... AGE.....

SCHOOL.....

DATE OF RECEIVING TREATMENT.	VASOCONSTRICTION NECESSARY.
.....	
.....	
.....	.....

OBSTRUCTION TO APPLICATION.

None.	Slight.	Medium.	Great.	Complete.

PATHOLOGY

LOCAL BOARD OF HEALTH, HINDMARSH.

Since it was decided to use vasoconstriction in every case, the entry "vasoconstriction necessary" became redundant.

Opposition.

As innovations in public health are open to controversy (and this investigation was no exception), a summary of the history of the preparatory period is given.

Opposition was encountered from the medical profession generally, mainly perhaps because the experimental nature of the undertaking was not fully realized; but my thanks are due to Dr. R. H. von der Borch, whose special rhinological knowledge was very helpful, and to Dr. D. Robson Wallman. Opposition was of such a nature at one stage that the alarm created nearly stopped the experiment. Noticing a cabled report that 5,000 children had been treated at Toronto, Canada, I communicated with the Minister for Health there, asking if any damage was occasioned by their treatment. I received an immediate reply: "No harmful effects nasal zinc sulphate treatment. 5,000 children." On the publication of this information, which was later followed by a letter, also published, most of the opposition disappeared. Throughout this investigation I worked with the full knowledge of the Advisory Committee on Infantile Paralysis appointed by the South Australian Government, which, because of the unproven value of the work, could not give public approval to it. Later, when the Deputy Minister for Health, Dr. B. T. McGie, Ontario, forwarded to me the report of Tisdall, Brown, Defries, Ross and Sellers,[5] it was immediately made available to the committee.

The Application of the Spray.

On the basis of evidence then available it seemed that the idea given by Peet, Echols and Richter,[1] that preliminary spraying with a vasoconstrictor and local anæsthetic would be advantageous, was worthy of trial, and it was decided to treat every child with a preliminary spray of a solution containing the following drugs:

Ephedrine	1·0%
Sodium chloride	0·5%
"Pontocaine"	1·0%

The actual application was by means of a De Vilbiss atomizer, number 156, with spray top number 156 N.C., introduced above the middle turbinate. It was realized that it might be an improvement if the head was placed sufficiently far back to allow small amounts of the fluid to accumulate at the top of the nose. This was achieved by putting the child's shoulders on a high, hard pillow and allowing the crown of the head to become the nearest point to the table. The object was to achieve a similar position to that subsequently illustrated in the article by Gutteridge[3] and by Shahinian, Bacher, McNaught and Newell.[4] The zinc sulphate solution contained the following drugs:

Zinc sulphate	1·0%
"Pontocaine"	0·5%
Sodium chloride	1·0%

Each atomizer was tested to ascertain the number of compressions of the bellows required to deliver one cubic centimetre of the fluid, and the required one cubic centimetre was thereby approximated in the actual treatment.

Spraying was commenced at Hindmarsh on December 22, 1937, soon after the commencement of the epidemic and was continued at intervals until February 3. Two hundred and fifty-one children were treated altogether. One hundred and twenty-one of these received two treatments, at an interval of approximately three weeks. It was found possible for three practitioners to treat up to 90 children in an hour. Treatment was continued as long as required, and ceased on February 3, 1938. One hundred and twenty-six of the 251 children had been immunized against diphtheria.

It was thought that the procedure might be difficult. In no case was the obstruction complete, although there was some degree of obstruction in seventeen cases. In three cases the obstruction was great or considerable, in two it was of moderate amount, while in the remainder it was slight. Treatment was thus possible in every case. The effect of the preliminary spraying of the ephedrine-anæsthetic solution was remarkable, and the shrinkage in the nasal mucosa most effective. It is interesting to note that Kramer, Grossman and G. C. Parker[6] have found that a mixture of adrenaline and ephedrine sulphate is an efficient deterrent to infection when sprayed into the noses of monkeys before instillations of poliomyelitis virus.

Incidence of Anosmia.

In order to determine by loss of sense of smell and the efficacy of the spraying, visits were made to about thirty children. The records showed that about three-quarters of these had anosmia during the first week. Unfortunately the records of this visiting cannot be traced; but 93 children were questioned on coming up for their second dose, and every effort was made to see that only reliable answers were entered. Ten infants, and those unable to give reliable answers, were counted as not having lost their sense of smell. By this method, as can be seen from Table I, there is a tendency for the greatest number of children to have anosmia for a period of three, seven or fourteen days.

TABLE I.

Number of Children.	Period of Anosmia. (days).
26	0
3	2
14	3
2	5
2	6
23	7
3	8
1	10
1	11
11	14
1	17
4	21
1	23
1	28
93	

When memory is concerned such aggregation of figures about a certain day cannot be taken as accurate. For example, the 23 people who state that they lost their sense of smell for seven days should be considered as probably having lost it for a greater period than three days, but not longer than seven days. This difficulty was realized at the time of questioning, and efforts were made to ascertain the actual day upon which they remembered recovering their smell. It will be seen that 67 children had some degree of anosmia for at least two days.

Sequelæ.

There were no serious after-effects in any case. A few children complained of headache and dizziness. A few slept particularly well the night after treatment, and some were reported as having voracious appetites. Sneezing within five minutes to half an hour after the spraying was common. Vomiting was not reported in any instance. All the children were well the evening after treatment, with one or two exceptions; these were free of trouble the next morning. Except for one adult not included in this series, in no instance did the loss of sense of smell last longer than 28 days, and in only six instances did it last for three weeks or longer.

One child, treated on December 23, was found to be suffering from poliomyelitis on December 29, and had been ill for twenty-four hours previously. It is practically certain that this child was in the incubation period when sprayed, and consequently this spraying could not have had any effect one way or the other. None of the other children developed the disease, despite the fact that the patient cited above slept in the same bed with three of them, and spent part of several days in the house of a group of four others. It is realized that the haphazard incidence of poliomyelitis might easily account for these results.

The Relative Insusceptibility to Poliomyelitis of Children Immunized against Diphtheria.

Quite early in the campaign rumours were circulated that children who had been immunized against diphtheria were not being affected by infantile paralysis. At first these remarks were not given serious attention. Towards the end of January, however, on looking up our index of immunized children, who constituted over 40% of the child population, we could find only three who had contracted the disease, whereas twenty of the unimmunized children in the same age group (the other 60%) were sufferers. It became known that in Glenelg and Brighton none of the immunized children contracted infantile paralysis. In these districts 813 and 516 respectively were immunized. When the total number of immunized children in these three districts was computed, it was found that four cases occurred among 2,603 children; that is, one child in every 650 became infected. On the other hand, in the group not immunized against diphtheria there were 22 cases among 4,197, or one child in 190. The situation later was found to be not so definite as this. Recently the Central Board of Health kindly furnished me with figures from other districts where a substantial proportion of the children were immunized, and it was found that of a total of approximately 19,853 children from these districts 6,742 were immunized. Sixteen contracted infantile paralysis, an approximate incidence of 1 in 420; while among the remaining 13,111 unimmunized children of the same age group, 45 cases occurred, an infectivity rate of approximately 1 in 290. Heaslip[7] found that among 238 cases of infantile paralysis, 21 children had been artificially immunized and nine had had diphtheria. Of these 30, eight gave a positive reaction to the Schick test. Such a rate of relapse to a positive reaction to the Schick test or lack of conversion to the "no-reaction" condition is far greater than I have met with in the Schick testing of 400 children after immunization. In fact, as reported of my work by Heaslip,[7] of 355 children who had been immunized in the town of Hindmarsh at least twelve months previously, two gave weak positive reactions, and these had received an incomplete dosage of anatoxin.

Calculation of Standard Deviation.

The groups at risk consisted of 6,742 immunized children and approximately 13,111 unimmunized children. The incidence of the disease in each group was 16 and 45 respectively. The total number of children and the total number of cases were respectively 19,853 and 61. The standard deviation (σ) was calculated according to the formula in the following manner.

$$\sigma = \sqrt{npq}$$

Where n = the number of persons
= 19,853, the sum of the immunized and and unimmunized.

p = the chance of any individual's becoming infected.

$$= \frac{61}{19853}$$

q = the chance of any individual's not becoming infected

$$= \frac{19792}{19853}$$

$$\sigma = 7{\cdot}8$$

The difference between the infected series
= (45 − 16)
= 29

Which is 3·7 times greater than the standard deviation.

The odds against the finding's being due to chance with the ratio (3·7) between this difference and standard error are greater than three thousand to one.

Summary.

1. The use of zinc sulphate solution as a nasal spray in the prevention of infantile paralysis is reported. No conclusion concerning efficacy of the method can be drawn from the small number treated.

2. The preliminary treatment with a solution of ephedrine and "Pontocaine" greatly facilitated the

procedure, and the recorded frequency (62·5%) of anosmia amongst those treated was higher than in many previous reports.

3. In towns where a substantial proportion of children were immunized against diphtheria, these children were significantly less likely to contract poliomyelitis than their unimmunized companions of the same age group.

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FRACTURES OF THE NECK OF THE FEMUR.

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AND

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AT the present time, when many surgeons consider that the use of the Smith-Petersen pin is the best, if not the only, method of treating fractures of the neck of the femur, we offer some observations based on a series of ten cases. These patients were treated by this method by various surgeons, and have come under our notice mostly within the last twelve months. At the outset we should like to point out that we do not want to belittle the method, but consider that any method must be judged not from its brilliant successes but from its average results. We think that advocates of this method have been deceived by the value of early mobilization of the joint and early weight bearing. May not these be a disadvantage? Is it not a fact that the advocates of the pinning operation and early function have overlooked the teaching of Thomas? Thomas contended

> . . . that fracture of the neck of the femur and fracture of the patella are analogous, in that they both produce unsoundness of the joint involved.

Thomas's aim was always to procure soundness of the hip joint—restoration rather than repair. To bring about this result he advocated prolonged immobilization and absence of weight-bearing. It is interesting to note that the workers at the McAusland Clinic, in a review of the treatment of fractures of the neck of the femur in 1934, made this observation:

> The limb should be kept immobilized for at least six months. The modern trend to cut short the period of immobilization in treating fractures near joints, and to institute early active motion to incite bony growth, is to be questioned. In our clinic we have tried both weight-bearing and non-weight-bearing methods, and from our experiences we conclude that the indiscriminate use of early motion of joints proximate to fractures is to be avoided. The rational view is to wait for healing and to put no strain on the site of fracture, until the surfaces have united sufficiently to bear the strain.

McMurray, in a recent publication, makes this comment:

> Another very serious drawback to the method [the pinning operation], and one that has only recently been recognized, is the disintegration of the small proximal fragment which follows the operation in a certain percentage of cases in whom the pinning has apparently been successful.

Watson Jones replied to this criticism in a subsequent issue of *The British Medical Journal* by stating that this complication occurred only in high cervical fractures, in which there was an avascular necrosis of the proximal fragment. He maintained that this state of the femoral head was the cause of the disintegration. That this is not the case is proved by the absence of the change after other methods of treatment, and further, by Case IV in our series. In this case the fracture line is still apparent in the X ray picture. It is a low cervical fracture, which never produces avascular necrosis of the proximal fragment.

It must be admitted that the three-flanged nail does give us a means of applying the principle of prolonged immobilization of the fragments; but we believe it has many drawbacks. The latter are complications of the method, and may be enumerated as follows: (i) complications associated with any operation or anæsthetic, especially in old people; (ii) osteitis due to the presence of the metal, (*a*) changes in the femoral head, (*b*) loosening of the pin; (iii) traumatic arthritis caused or aggravated by the impaction at operation; (iv) non-union or unsound union; (v) refracture, with fracture of the nail; (vi) damage to (*a*) the femoral head (for example, splitting of the bone) and (*b*) the acetabulum; (vii) trauma to an unsound joint caused by too early mobilization.

The following cases will demonstrate some of these conditions.

Clinical Records.

CASE I.—The patient, a male, aged forty-six years, fractured the neck of his left femur in June, 1936. He was treated between sandbags for fifteen weeks in the country, and then was removed to the city, where he was admitted to hospital. At that time he had non-union at the site of fracture, and at the left hip joint a deformity of flexion, adduction and external rotation. The following week a

Smith-Petersen pin was inserted by the open method. A subsequent X ray film showed the pin and fragments in good position. Four months later he complained of pain in the left hip, and an X ray photograph taken a few weeks later showed the pin protruding beyond the articular surface of the head of the femur and apparently extending into the acetabulum. A few days later a small incision was made over the head of the pin and the latter was withdrawn slightly. The following week the wound was infected. The subsequent history is that of septic arthritis of the left hip joint followed by septicæmia.

The main interest of this case is the onset of infection months after the insertion of the pin and following such a simple procedure as withdrawal of the pin for a short distance.

CASE II.—A female, aged fifty-nine years, fractured the neck of her right femur in August, 1937. A Smith-Petersen pin was inserted a fortnight later by the closed method with radiographic control. Eleven days later she had a rigor, and, as she had previously suffered from malaria, it was hoped that this was the cause of the rise in temperature. Ten days later, however, there was a thin sero-purulent discharge from the wound. X ray examination a month after the onset of the infection revealed no changes in the bone; but two months later there was a pathological dislocation of the right hip joint. There was still no evidence of involvement of the bone. All this time the patient was suffering much pain and at times was quite abnormal mentally.

A month later, that is, four months after the original operation, the pin was removed. A fortnight after this, X ray examination showed the fragments in good position; but there was no evidence of bony union and there was still no evidence radiographically of infection of the bones or joint. At the present time the patient is not suffering a great deal of pain, but she is finding it very difficult to move about wearing a caliper.

The condition of this woman's mouth was very bad, and it is possible that this had something to do with the infection.

CASE III.—A male, aged seventy-one years, fractured the neck of his right femur in January, 1935. A Smith-Petersen pin was inserted a fortnight later. He was discharged from hospital three months later and told to use his leg as if there was nothing wrong with it. The hip became painful very shortly afterwards. Rest gave him relief. Nine months after operation the pin was removed and he was told that firm union was present. A week later the hip became painful, but he persevered with movement and attempted to walk a fortnight after the pin had been removed. Two months later he was informed that non-union was present and a caliper was ordered. When seen in June, 1937, he still had non-union and was very anxious to be rid of his appliance, but declined to undergo a McMurray osteotomy. He was still having pain in the hip, especially after walking. In short, he was suffering not only from an ununited fracture, but also from an unsound hip joint.

CASE IV.—A female, aged fifty-eight years, had a Smith-Petersen pin inserted into the neck of her left femur in January, 1937. She was discharged from hospital after only three weeks and told to use her leg freely. At this time she described her symptoms as "mortal pain" in the hip, and the more she walked the worse was the pain. Because of the persistence of this pain the pin was removed three months after being inserted. Four months after operation her signs and symptoms were those of a subacute arthritis of the affected hip joint, but at no time was there any elevation of temperature. A Thomas's hip splint was applied and afforded her relief. Skiagrams were taken periodically, and it was not until twelve months after operation that the destruction of the head of the femur was visible. In this case the fracture was of the low cervical type, and at no time was there any sign of avascular necrosis of the femoral head.

CASE V.—A female, aged seventy-nine years, fractured the neck of her right femur in March, 1937. A Smith-Petersen pin was inserted a week later, but five months afterwards it was removed because of incipient damage to the hip joint. Her relatives stated that she had been in pain more or less constantly since the operation. In February, 1938, she was admitted to hospital again for an attempt to relieve her symptoms. The skiagram revealed non-union of the fracture with riding up of the distal fragment, as well as evidence of destructive changes in the hip joint.

This patient had been told, after removal of the pin, that perfect union had taken place, and evidence to this effect had been given in a court of law.

CASE VI.—A female, aged seventy-three years, had a Smith-Petersen pin inserted about two years before she was first seen by us, when she was complaining of severe pain in the left hip of several months' duration. It appears that the first pin had been inserted too far, and it was necessary to remove and replace it. The skiagram revealed pronounced destruction of the head of the femur, and what appeared to be a wandering acetabulum. The pin was still *in situ*. It was removed, and extension was applied to the limb with great relief. Four months later she was able to walk with a caliper.

CASE VII.—A male, aged forty-one years, fractured the neck of his left femur in October, 1936. A Smith-Petersen pin was inserted four months later. He was discharged from hospital three months after operation, wearing a caliper. At this time the X ray film revealed the pin and fragments in excellent position, but sclerosis of the fragments was evident. In September, 1937, he was able to drive a motor car, walk any distance and do everything he wanted to. The range of movement at the hip joint was very good, while forcing any movement did not cause pain. The X ray picture still revealed non-union with sclerosis of the fragments. He took part in the last Anzac Day march, and completed the route with ease. An X ray picture taken a few days later revealed the same state of affairs as on previous occasions.

This case must be considered to have had a good result. To us the main point of interest is the fact that the patient has a sound joint. This latter fact may be due to the four months' immobilization he had before he came to the city and had the pin inserted; for after the operation movements were instituted within ten days. Moreover, it does show that non-union with a pin in position does not cause disability *per se*, and it is a good example of the truth contained in the principles laid down by Thomas over fifty years ago. This case is included mainly to demonstrate the importance of the state of the hip joint after any injury in its vicinity.

CASE VIII.—A female, aged seventy-five years, had a Smith-Petersen pin inserted into the neck of her left femur in December, 1936. She left the hospital eight weeks later. Ever since she has had pain in the left groin, the left sacro-iliac region and the outer side of the left thigh. All movements at the left hip joint are very limited; there is no shortening. The skiagram revealed incomplete union at the site of fracture, as well as some irregularity of the acetabulum.

This patient must be looked upon as having an unsound joint.

CASE IX.—A female, aged forty-eight years, fractured the neck of her left femur in April, 1935. A Smith-Petersen pin was inserted a week later. In October of the same year the radiological report was to the effect that the fragments were in good position and bony union appeared to be well

advanced. In November, 1936, the patient developed lobar pneumonia, was admitted to hospital and died. An enthusiastic and inquisitive resident medical officer decided to investigate the state of the left femoral neck and hip joint. His dissection disclosed an eburnated and irregularly shaped femoral head, separated from a small length of femoral neck by a gap of 1·9 centimetres (three-quarters of an inch); this gap was bridged by the Smith-Petersen pin.

As far as we could learn from her relatives, this woman was able to walk about her house quite comfortably until the day she was removed to hospital with pneumonia.

Case X.—A male, aged sixty-two years, had a Smith-Petersen pin inserted into the neck of the left femur in January, 1935. The radiological report in July of the same year was to the effect that union appeared firm, with the fragments in good position. In February, 1936, while alighting from a tram, he fell to the ground and was unable to get up again, because of the pain in his left hip. He was taken to hospital by ambulance, and an X ray photograph revealed a refracture at the site of the original fracture of the femoral neck, as well as a fracture of the pin at the same site.

Comment.

Of the above complications only a few call for further comment.

Aseptic Osteitis.

The changes seen radiographically in the head of the femur in some of the cases suggest aseptic osteitis. We cannot recall having seen similar changes in any case after treatment by the other methods in use. The radiological appearance is somewhat similar to that seen in Perthes's disease of the hip joint and has nothing to do with the avascular necrosis of the proximal fragment that is so regularly seen in high cervical fractures. In Case IV of the above series the fracture is in the distal portion of the femoral neck, and in fractures at this site necrosis of the proximal fragment is rarely, if ever, seen. We have not been able to secure a specimen to have it examined pathologically, but the radiographic appearances are those of osteitis. This condition is probably due to the disturbance of the blood supply to the head of the femur. Why it should occur in some cases and not in others is difficult to understand. We have examined several possibilities and append some of them.

The concussion and trauma caused by hammering of the nail into the head fragment may be the primary cause; but against this is the fact that similar changes have not been reported after the fibular graft which is so popular in Holland. In that country fracture of the femoral neck is a very common injury and is not confined to people of middle age or older. This is due to the method of transport used by the greatest majority of the population, the bicycle.

Another possibility is the damage to the head at the time of the accident; but similar changes have not been reported after the other methods of treatment in use. The same argument pertains to the postulation of some constitutional defect as the cause.

All the evidence at our disposal points to the presence of the metal *plus* some unknown factor as being the cause of the osteitis. Unfortunately, in the present state of our knowledge it is not possible to say what this unknown factor is. Neither of us can recall having seen the changes described above after the use of the Whitman method of treatment. They were not seen over a period of twenty-five years in the McAusland Clinic, where the Whitman method is still the one in use in the majority of cases. Phillip Wilson, of New York, told one of us in August, 1936, that he was seeing quite a number of his earlier patients, whose fractures he had treated in the clinic by means of Smith-Petersen pins. They had painful hips and changes in the femoral head resembling those of Perthes's disease. He went on to say that he wondered whether he would be inserting pins in fractures of the femoral neck at the end of another two years. The radiographic changes do not matter so much as the pain associated with those changes: for it means a further period of enforced rest followed by the wearing of an appliance until the joint becomes sound, to use a phrase that meant so much to Thomas.

From all the information we can obtain there is no definite period after the insertion of the pin at which this complication of osteitis occurs. As time goes on, more of these patients will probably be seen.

Traumatic Arthritis.

Traumatic arthritis may result from the injury. As Thomas pointed out, there must be a certain amount of joint involvement in every case of fracture of the femoral neck proximal to the intertrochanteric line, as a study of the capsular attachments to the upper end of the femur will prove. If there is any damage to the articular cartilage at the time of the injury, it is reasonable to assume that the impaction and driving in of the pin at operation will aggravate the condition. In such cases early mobilization will be detrimental, as the joint is in an unsound state; rest is the only form of treatment for such joints. We believe that failure to apply this principle is the cause of the increasing limitation of movement in so many cases after the use of the Smith-Petersen pin.

Non-Union and Unsound Union.

We should not rely on the X ray picture for diagnosis or differentiation between non-union and unsound union, for the skiagram can be very misleading as to the state of union in the neck of the femur. It is possible that most of the cases labelled "incomplete union" on X ray evidence are in reality cases of non-union. Whitman has recently pointed out that during the repair of a fracture decalcification combined with softening occurs in the fragments contiguous to the fracture site. For repair to take place with certainty a collapse of these softened areas must occur, so that the healthy and more normal bone will come together. This absorption does not invariably take place, but it is of frequent

occurrence in bone in which there is much cancellous tissue, and is seen in people, especially females, over the age of forty years. This explanation accounts for quite a number of the deformities that follow fractures near the ends of long bones, in cases in which the treatment has been carried out adequately and assiduously (for example, Colles's fracture). In the neck of the femur the Smith-Petersen pin will prevent the fragments' coming together if this absorption occurs, and may tend to increase it by setting up and prolonging the hyperæmia; in such cases non-union will be the result. If the pin is removed before union is complete or before it has even occurred, the percentages of non-unions will naturally increase, while, if the pin is left in too long, the chances of the development of aseptic osteitis in the head of the femur are probably very much greater.

Stirling has done some very interesting work in regard to the changes brought about in bone by the presence of metal. Normally, after a fracture, the tissues in the vicinity undergo an alteration in the pH value to the acid side. This reaction remains acid for ten days or so and plays a large part in the subsequent repair of the damaged bone. If metal is used, this increase in acidity is prevented, and there is a consequent delay in the repair of the fracture. In his article Stirling advises strongly against the use of metal in any form in the fixation of fractures, but makes an exception of the Smith-Petersen pin. Why he makes this exception he does not state.

Conclusions.

We reiterate that we have seen some brilliant results from the use of a Smith-Petersen pin; but we consider that in the above series of cases there was sufficient evidence of interference with function to lead us to believe that the Smith-Petersen pin was not the ideal method of treatment. Moreover, we are convinced that during the next few years the limitations of the method will be appreciated.

We have suggested some of the possible causes of these unsatisfactory results, and we think that a larger series of cases, observed over a longer period of time, will enable us to be more definite.

ACCIDENTAL HÆMORRHAGE.

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THE cases reported here were of severe accidental hæmorrhage. Rigby, as early as 1775, described the condition of accidental hæmorrhage. In 1912 Couvelaire[1] presented a clearer picture of this acute severe form. The name of utero-placental apoplexy, which Couvelaire gave to the condition, aptly describes the sudden collapse of a pregnant woman and rapid death.

REPORTS OF CASES.

Case I.

J.M.K. was a woman of thirty-eight years. She was 32 weeks pregnant in her ninth pregnancy. Her eight children were all alive, the eldest being sixteen years and the youngest three years of age. The heaviest at birth was 4·1 kilograms (nine pounds). All her labours had been easy and the puerperia normal. She had had eclampsia with the second. At 28 weeks' gestation she attended the out-patient department. The urine contained no albumin, her systolic blood pressure was 120 and diastolic 80 millimetres of mercury. The fœtus was in the right occipito-anterior position and the fœtal heart sounds were audible.

At 4.30 p.m. on September 21, 1937, she was admitted to hospital. She complained that for two and a half hours she had had abdominal pains which she took to be labour pains, and she had had an aching pain over the sacrum for four hours. She had no bleeding *per vaginam*. On examination she was seen to be very pale and sweating. The uterus was enlarged to a 34 weeks' pregnancy and did not relax as in normal labour, but remained tense, causing continuous pain. There were areas of great tenderness over the surface of the uterus, the whole organ being more sensitive than usual. The fœtus could not be outlined and no heart sounds could be heard. At vaginal examination the cervix admitted two fingers and the cervical canal measured under 1·3 centimetres (half an inch), conditions to be found in many *multiparæ* not in labour. The membranes were tense and bulging and the presenting part was high up and ill defined. No placenta was felt. A slight degree of œdema was present over both tibiæ and ankles. The pulse rate was 100 per minute and the pulse volume was reduced. The systolic blood pressure was 85 and the diastolic 60 millimetres of mercury. The urine contained no albumin.

It was decided to watch the patient to see if the uterus would expel its contents, the diagnosis being partial separation of the placenta with concealed bleeding. The abdominal pain continued and the uterus remained tense. The patient became exceedingly pale on occasions without accompanying symptoms, and the pulse rate was 80 beats per minute throughout and following the period of pallor. The pulse rate ranged between 80 and 100 beats per minute. These paroxysms of pallor with the slow pulse are most unusual.

After two hours' observation the tenderness to palpation had spread up to the costal margins and the uterine tumour had altered in shape. This suggested that partial rupture of the uterus was occurring. The pulse rate remained at about 100 beats per minute, but the abdominal pain increased and the patient generally appeared worse. Abdominal section was decided after three and a quarter hours' observation. No bleeding had occurred *per vaginam*.

The operation was performed under open ether anæsthesia. The abdomen was opened by a lower mid-line incision 10·0 centimetres (four inches) in length. The peritoneal cavity contained about 0·6 litre (half a pint) of sanious fluid. The uterus was enlarged to the size of a 32 weeks' gestation and was in a state of firm distension. The muscle was mottled with many small blue areas, most thickly distributed over the front of the uterus and over its lower posterior aspect. These areas are due to vascular degeneration followed by small hæmorrhages amongst the muscle fibres. The appearance of the muscle fibres is one of toxic spoiling passing on to degeneration. This is the cause of the lack of contractions in the uterus.

There were three shallow irregular fissures on the surface of the uterus, two on the anterior surface and one over the lower segment in the pouch of Douglas. These were the points of impending rupture. The uterus was opened by the classical incision. The fœtus was removed stillborn. The placenta lay on the posterior wall of the uterus and was completely separated by old and recent blood clot from the placental site. The placenta and clot were easily removed.

The uterus contracted and retracted very little. The cavity of the uterus was more ragged in appearance than usual. The muscle was blue and friable. One cubic centi-

metre of pituitrin and one cubic centimetre of "Ergometrin" were injected into the deltoid muscle, and hot sponges were applied to the uterus. After about five minutes' observation the uterus remained relaxed and there was some bright red blood within the uterine cavity. This determined a supravaginal hysterectomy. The Cæsarean incision in the uterus was closed with three through-and-through sutures and hysterectomy was performed. The patient's condition at this stage of the operation was very poor; the pulse could just be felt at the wrist. Blood transfusion was commenced and continued after the operation was completed, 450 cubic centimetres of blood being given.

In the ward the patient gradually improved and made an uneventful recovery.

Comment.

This patient's slow heart rate, between 80 and 100 beats per minute, during the condition of shock and hæmorrhage is rarely seen and could be misleading if the other signs were ignored.

Case II.

The patient, M.B., aged twenty-two years, was a *primipara*. Her previous health had been good. She had had measles as a child and an appendicectomy three years earlier. She had suffered from vomiting during the first three months of pregnancy; this had been associated with retroversion of the uterus, which was replaced.

The patient was admitted to hospital at 9.30 a.m. on April 9, 1937. She complained of having suffered from a dull aching pain over the sacrum for twelve hours. About eight hours before admission to hospital she had vomited. After this the sacral pain had spread round to the abdomen and bleeding *per vaginam* had commenced. The bleeding which followed had saturated five bath towels and the abdominal pain had gradually become worse.

On examination the patient was seen to be pale. The uterus was enlarged by a gestation which had reached the thirty-second week. This size corresponded with the calculated duration of pregnancy from the last menstrual period. The fœtus was palpated and found to lie in the left occipito-posterior position, with heart sounds audible in the left flank. Strong first-stage uterine contractions were present, the muscle's tone remaining high during the relaxation period. Uterine tenderness was present but not severe. Examination *per vaginam* revealed that the external os admitted one finger, the cervical canal was about 2·5 centimetres (one inch) in length, and the membranes were intact. A small head was presenting in the upper part of the pelvis. There was no blood in the vagina at that time. The patient's pulse rate was 116 beats per minute and had a moderately good volume. The systolic blood pressure reading was 140 and diastolic 83 millimetres of mercury. The urine test revealed albumin, the coagulum settling to half the volume of urine tested. The patient was not œdematous.

A diagnosis of mild accidental hæmorrhage with albuminuric toxæmia was made. In the presence of what appeared to be good uterine contractions and the absence of external hæmorrhage no active treatment was undertaken.

After one and a half hours' observation the pulse rate remained unaltered. The uterus had enlarged slightly without external bleeding, and the woman had vomited several times. In the next one and a half hours the pulse rate rose to 128 beats per minute, and the uterus had enlarged to the size of a 36 weeks' gestation, still without external bleeding. As the uterus enlarged the uterine contractions became less prominent and the uterine tenderness more severe.

In another half-hour bleeding *per vaginam* occurred in a small amount. In a further half-hour the uterus reached the size of a 38 weeks' gestation. Vaginal examination revealed that no progress of the labour from the first vaginal examination had been made. The pulse rate remained at 128 beats per minute and the general condition was unchanged. In view of the rapidly increasing concealed hæmorrhage and the lack of progress of the labour, Cæsarean section was decided upon while the patient's general condition would permit the operation without undue risk.

Four and a half hours after her admission operation was performed under open ether anæsthesia. When the abdomen was opened the peritoneal cavity was found to contain about half a pint of blood-stained fluid. The uterus was tensely distended to the size of a 38 weeks' gestation. All over the surface of the uterus the small hæmorrhagic spots were thickly distributed—so thickly as to be confluent in several areas. The classical Cæsarean incision ten centimetres (four inches) long was made to open the uterus. A fœtus equivalent in size to that of gestation of 32 weeks, detached placenta and much blood clot delivered spontaneously through the wound. The cavity of the uterus presented a ragged appearance with bundles of degenerate muscles. Pituitrin and "Ergometrin" in a dose of one cubic centimetre each were injected into the deltoid muscle, and a hot pad was applied to the uterus. After about five minutes' observation no contractions of the uterus had occurred. Hysterectomy was thought to be the safest line of treatment. The Cæsarean wound was closed and a supravaginal hysterectomy rapidly performed. At the end of the operation the patient's pulse rate was 160 beats per minute. Blood transfusion was performed, 650 cubic centimetres of citrated blood being given. One and a half hours after the operation her pulse rate had fallen to 130 beats per minute. She made an uninterrupted recovery and was discharged on the twenty-second day after operation.

Comment.

The extent of the degenerative changes in this uterus was a surprise. When the woman was first seen the uterine action appeared to be sufficient to complete the delivery unaided; but the condition of the uterine wall, revealed at operation, showed the difficulties to be encountered in attempts to predict the course of accidental hæmorrhage.

Case III.

The patient, who was aged thirty-four years, had been pregnant ten years before. This pregnancy had been terminated by the instrumental delivery of an infant weighing 5·0 kilograms (eleven pounds). She suffered from "kidney trouble" before and *phlegmasia alba dolens* after her first pregnancy. Six years before admission she had suffered from "kidney trouble". With this pregnancy hyperemesis was present at the second month.

She was admitted at 2.15 p.m. on February 17, 1937. She complained of some bleeding *per vaginam*, pain over the whole abdomen and aching over the sacrum, which had existed for the previous four hours. There had been some epigastric pain the night before. For two weeks there had been slight swelling of her feet.

On examination the woman's face was seen to be pale and her lips pink. The uterus was enlarged to the size of a 28 weeks' gestation. This size corresponded to the period of gestation calculated from the last menstrual period. The uterus was firm with areas of tenderness. No attempts at true relaxation were made. The fœtus could not be outlined with certainty and the fœtal heart sounds were not heard. Slight bleeding *per vaginam* was occurring. The pulse rate was 140 beats per minute, and the volume of the pulse was moderately good. The systolic blood pressure reading was 140 and the diastolic 110 millimetres of mercury. Œdema was detectable over the feet and the test for albumin in the urine revealed a faint coagulum.

Accidental hæmorrhage was the diagnosis made. The treatment was expectant, an abdominal binder alone being applied.

The pulse rate continued at between 130 and 140 beats per minute and the uterus continued to be tense. After four hours the bleeding *per vaginam* increased, determining a vaginal examination. The cervix was dilated to two fingers' breadth and was taking up. The membranes were intact across the internal os and the presenting part was high up. The examination increased the bleeding and it was necessary to plug the vagina with cotton-wool plugs and attach a perineal binder to the abdominal one. This was done under gas and oxygen anæsthesia. This

bleeding caused the pulse volume to diminish although the rate did not rise. Three hundred cubic centimetres of gum-saline solution were given intravenously in the next hour, but the patient's general condition became worse and the pulse rate rose to 150 beats per minute. Then 500 cubic centimetres of citrated blood were given intravenously, a further hour being taken up. The pulse rate now was 160 beats per minute and the vaginal plugging had become soaked through with blood. Operation was decided upon in view of the rising pulse rate and the inability of the uterus to contract normally. This was seven hours after admission.

When the peritoneal cavity was opened at operation the uterus, enlarged by a 28 weeks' gestation, presented. There was a small amount of sanious fluid free in the abdomen. The uterus was opened by the classical incision and a dead fœtus corresponding in size to one of 22 weeks was removed. The placenta was situated on the lower posterior wall of the uterus and was almost completely separated by blood clot. The placenta and clot were removed. Degenerate hæmorrhagic areas were seen to be scattered through the uterine muscle, being most dense over the placental site. After the intramuscular injection of pituitrin and "Ergometrin" and the application of a hot sponge to the uterus as in the previous cases, no contractions of the uterus occurred and bleeding continued within its cavity. At this stage of the operation the pulse could not be felt at the wrist. In spite of this, supravaginal hysterectomy was performed. Five hundred cubic centimetres of normal saline solution were poured into the peritoneal cavity as the abdomen was being closed. At the end of the operation the pulse was just perceptible at the wrist. Three hundred and fifty cubic centimetres of citrated blood were then given intravenously, and the pulse rate fell to 140 beats per minute and the volume likewise improved. The next morning the pulse rate was 110 beats per minute. The patient was discharged on the twenty-second day without post-operative complications.

Comment.

Conservative measures were unsuccessful in this case. The plugging of the vagina actually added to the risk of sepsis, and the woman would certainly have died undelivered if operation had not been performed. The risk of the operation was greatly increased by the patient's being allowed to pass so far into the condition of shock and hæmorrhage. The patient's power of recovery, aided by blood transfusion, was truly remarkable.

Case IV.

The patient, C.G., was a *primipara*, aged twenty-three years. She had suffered no relevant previous illness. She was admitted to hospital at 10 a.m. on May 20, 1937. In a letter her doctor stated that he had seen her first fourteen hours before, when she was thought to have had a "show" without labour pains. A vaginal examination had shown that the cervix was not dilated and the head, the presenting part, was high up. The pulse rate was 80 beats per minute. When she was reexamined three hours later the cervix had commenced to dilate and the hæmorrhage had ceased. Morphine in a dose of 0·01 gramme (one-sixth of a grain) had been administered, producing a good night's sleep. On the morning of admission moderate bleeding *per vaginam* had recommenced; the pulse rate was 100 beats per minute and the uterus was large and tender.

On admission the patient had stated that she had noticed an aching pain in the back (sacral region) twelve days before; this had disappeared, to return six days before, and again during the two days prior to admission. Bleeding *per vaginam* had occurred the night before admission in a small amount and pain in the lower part of the abdomen had begun about five hours before admission. On examination the woman was seen to be very pale. The uterus was enlarged to the size of a full-time pregnancy; calculating from her last menstrual period she was one week overdue. The uterus remained in a tense state and was tender all over to palpation. This made it difficult to outline the fœtus, which seemed to be about full-time with the head engaged in the pelvis. Fœtal heart sounds could not be heard. Vaginal examination showed the cervix to be dilated to two fingers' breadth and the cervical canal had been taken up. No placenta was felt. The fœtal head was engaged through the brim and the examining finger was blood-stained. The pulse rate was 120 beats per minute, the volume being reduced. The systolic blood pressure reading was 118 and the diastolic 65 millimetres of mercury. Albuminuria was present.

The diagnosis of accidental hæmorrhage was made, most of the hæmorrhage being concealed. A consideration of the tense condition of the uterus with concealed hæmorrhage, the ineffective labour contractions and distension of the uterus, the rising pulse rate and the presence of mild toxæmia determined the performance of Cæsarean section while the patient was in a fit condition.

One and a half hours after admission the operation was performed. The abdomen was opened and the enlarged uterus presented in the wound. The uterus was opened and a full-time living child delivered. The placenta was situated low on the posterior wall of the uterus. It had partially separated and blood clot had raised the membranes above the separation. The exact fraction of the placenta separated was not observed. After the removal of the placenta the uterus contracted in spite of the small bluish areas of degeneration, which were mainly over the placental site. Pituitrin and "Ergometrin" were injected into the deltoid, and the uterus became quite firm. The wound in the uterus was closed and the abdominal wound was closed. The pulse rate following the operation was 145 beats per minute. A blood transfusion of 500 cubic centimetres of citrated blood was given. This reduced the pulse rate to 120 beats per minute and improved the pulse volume.

The patient's convalescence was uneventful. She went home on the twenty-seventh day.

Comment.

This case shows that although dark areas of degeneration are present in the uterus, the removal of the organ does not necessarily follow. The over-distension with the extravasated blood was the cause of its non-contraction in the early part of the labour, but after the removal of the fœtus and placenta there were sufficient undamaged muscle fibres to contract and control the hæmorrhage from the uterus.

Case V.

The patient, O.S., was in her seventh pregnancy, at the age of forty-two years. The oldest child was twenty-one and the youngest two years of age. All these pregnancies had been passed through without abnormality, the heaviest child being 4·6 kilograms (ten and a quarter pounds) at birth and the longest labour five hours. She had had two miscarriages, the last three years before. She had suffered no previous illness.

Six weeks before admission to hospital she had attended the out-patient department, complaining of irritation of the vulva. At that time the uterus was enlarged to the size of a 30 weeks' pregnancy. The fœtus lay in the left occipito-anterior position and heart sounds were heard. The systolic blood pressure reading was 120 and the diastolic 80 millimetres of mercury. No abnormal substances in the urine were revealed by the tests. These findings are within normal limits.

The patient was admitted at 6·5 a.m. on May 4, 1937. She stated that pains had begun over the sacrum and across the lower part of the abdomen one hour before. These pains were similar to her previous labour pains. On examination the woman was seen to be pale; the uterus was enlarged to a 38 weeks' pregnancy and regularly occurring contractions were present. The exact position of the fœtus could not be determined, but the head could be felt engaging in the brim. No fœtal heart sounds were heard. No bleeding *per vaginam* had occurred. The systolic blood pressure reading was 170, the diastolic 130 millimetres of mercury. The test for albumin in the urine gave a coagulum settling to one-third of the volume tested. It was thought at this stage that the labour would terminate rapidly and that the severe albuminuric toxæmia had caused the death of the child. A tight abdominal binder was applied.

After two hours the patient's condition had not changed and the labour had not advanced. In a further period of two and a half hours the patient gradually became hot and sweating. The uterus was more firmly contracted and relaxation could not be easily detected. A vaginal examination revealed the cervix to be dilated two fingers' breadth and the cervical canal to be 0·6 centimetre (a quarter of an inch) in length. The membranes had ruptured and the head was presenting at about the level of the middle of the pelvis. No bleeding was present. The pulse rate had risen to 110 beats per minute, the volume being unaltered. The urine tested now was solid with albumin. Blood examination at this point revealed 3,650,000 red blood cells per cubic millimetre and a hæmoglobin value of 52%.

The lack of progress of the labour and the rising pulse rate and the increasingly tense uterus all pointed to a concealed accidental hæmorrhage; but it was still thought that as this was the seventh labour and that the previous labours had been rapid, the cervix would dilate rapidly and no interference would be necessary. So attention to the patient was still confined to observation.

Eight hours after admission the patient was becoming more anxious and fatigued. The pulse rate had risen to 130 per minute. No external bleeding had occurred. The uterus had become tense and firm. Operation was thought to be the best course to pursue.

A classical Cæsarean section through a mid-line incision was performed under open ether anæsthesia. The uterus presented in the lower part of the wound. There were several ounces of sanious fluid in the peritoneal cavity. The uterus did not show a great number of dark areas of spoiling on its surface. The uterus was incised and an almost full-time stillborn child removed. The placenta was situated at the fundus and was partially separated by fluid blood and recently formed clots. This was manually removed. The uterine muscle became partially active on the injection of pituitrin and "Ergometrin" into the deltoid and stimulation by heat. It was thought sufficiently active not to require removal. The wound in the uterus and the abdominal wound were closed. Throughout the operation the patient's pulse was not good, ranging in rate from 140 to 150 beats per minute and having a small volume. As the abdomen was being closed the pulse became imperceptible at the wrist. Injection of gum-saline solution was commenced; 450 cubic centimetres were given. This had practically no effect in reviving the condition of the cardiovascular system. Adrenaline and "Coramine" were of no avail. The patient died about 20 minutes after the completion of the operation, before a blood transfusion could be administered.

Comment.

To begin with, this patient was normally a small, pale, thin woman and multiparous. Her reserves to withstand shock and hæmorrhage were at a minimum. The areas of degeneration to be seen in the uterus were the least in all this series of cases. The two alternative courses that might have saved her life were earlier operation and the performance of blood transfusion before, during and after the operation.

Case VI.

The patient, M.A., was thirty-three years of age and she was pregnant for the sixth time. Her five children were born without abnormality, except the last, one year previously, when albuminuria and *ante partum* hæmorrhage had occurred. But these abnormalities had not been sufficiently severe to cause the death of the child. The woman had previously suffered from pneumonia and had had her appendix removed.

Eight weeks before admission to hospital the patient attended the out-patient department, when she was found to have a uterus the size of a twenty-two weeks' gestation. The urine tests revealed no abnormal substances. The systolic blood pressure reading was 125 and the diastolic 55 millimetres of mercury. Her teeth were badly carious. She did not attend the out-patient department again before admission.

She was admitted at 5.15 a.m. on September 9, 1937. She stated that pains, which she took to be early labour pains, had begun three hours before; they were mainly over the sacrum. She had had no bleeding *per vaginam*. Fœtal movements had not been felt in the last twelve hours. On examination the woman was seen to be pale. The uterus was enlarged to the size of a 34 weeks' pregnancy. The stage the pregnancy had reached, calculated from the last menstrual period, was 30 weeks, and the estimation from her visit to the out-patient department was 30 weeks. These estimations were misleading, as the fœtus proved to be of about 34 weeks when delivered. Weak uterine contractions were present. The fœtus lay in the left occipito-anterior position, with the head engaging in the brim of the pelvis. No fœtal heart sounds were heard. The pulse was about 100 beats per minute in rate and its volume was normal. The systolic blood pressure reading was 95 and the diastolic 55 millimetres of mercury. The urine tests revealed no abnormality. The patient was thought to be in early premature labour. No treatment was called for.

After three hours a little bleeding occurred *per vaginam*. The uterine contractions were increased in power, the uterus remaining a little hypertensive. In half an hour the pulse rate rose to 120 beats per minute. The systolic blood pressure reading then was 95 and the diastolic 55 millimetres of mercury. Examination of the urine in another half-hour showed a faint coagulum of albumin. The half-hourly pulse readings until the reading of 120 beats were as follows: 96, 116, 116, 120 beats per minute; reduction in volume occurred, corresponding to the higher rates. At the time of this last reading the uterus had become very tense and slightly tender. Labour contractions had ceased. A blood count taken at this time showed 4,730,000 red blood cells per cubic millimetre and a hæmoglobin value of 48%.

At this stage Cæsarean section was decided upon, on account of the rise in pulse rate without external bleeding, the tense condition of the uterus, the absence of true labour contractions, and the enlargement of the uterus beyond the period of gestation, calculated from the last menstrual period and her visit to the out-patient department (this proved misleading). The diagnosis was accidental hæmorrhage, mostly concealed.

Six and a half hours after admission to hospital operation was performed under open ether anæsthesia. When the lower part of the abdomen was opened the enlarged uterus presented in the wound. The uterine wall contained few areas of black toxic degeneration and the muscle appeared to have fairly good contractile power. When the uterus was opened the placenta was encountered on the anterior wall, almost wholly separated by fresh blood clot. A dead fœtus of 34 weeks was delivered through an incision in the placenta. The placenta and membranes were then removed. The uterus contracted and retracted readily. The wounds in uterus and abdomen were closed. The patient's general condition was such as to give no anxiety. Blood transfusion was not needed. The patient was discharged on the twenty-third day, having had slight pyrexia for a few days after the operation.

Comment.

In retrospect this case was the least deserving of Cæsarean section in this series. On the assumption that as a result of concealed hæmorrhage the uterus was one month larger than would ordinarily have been expected, the operation was performed early to give the patient her best chance of surviving. The difficulty of estimating the amount of the intrauterine hæmorrhage is well shown.

DISCUSSION.

Most authorities agree that of the many obstetric complications the severe accidental hæmorrhages are among the most difficult to treat. Broadly speaking, the treatment is directed against shock and hæmorrhage and towards aiding the uterus to expel its contents as soon as possible. In this particular series of cases the patients suffered from the most severe form of accidental hæmorrhage. They were treated by abdominal section. The occurrence of such cases is rare.

Albuminuric toxæmia formed the background in each case. This may be present in a mild form, showing only as slight œdema over the tibia, and perhaps a raised blood pressure, for some weeks before the acute symptoms appear. The onset of acute symptoms was relatively sudden, the longest warning of sacral pain occurring in Case IV twelve days before the onset of the more acute symptoms. The acute symptoms were of twelve hours' duration at most on admission to hospital. The abdominal pain was mistaken for labour pains, and the severe sacral aching was complained of persistently in each case.

The general condition of the patients was due to a combination of shock and hæmorrhage, the shock being greater than one would associate with a hæmorrhage of a similar amount from another cause. Apparently the shock is mainly caused through the distension and irritation of the uterus. The pulse was used as the simplest indicator of the change in the patient's general condition. The reaction of the pulse rate to hæmorrhage is variable. As an average, when the rate of 120 beats per minute has been reached, the mechanism for the adjustment of the volume lost has attained its limit. A relatively small further loss causes the breakdown of the mechanism, and the pulse rate will rise rapidly to an uncountable level. In this series a rate of 120 beats per minute, or more, was regarded as one of the indications for operation. The cardio-vascular system as a whole is rendered unstable by toxæmia. Evidence of this was pronounced in Case V; probably it was a big factor in the patient's sudden collapse. In Case I the pulse rate was unexpectedly slow, ranging from 80 to 100 beats per minute; the skin was very pale throughout the stages of blood loss.

A blood pressure raised by the toxæmia will fall with blood loss. When the patient is seen in the acute stages the blood pressure may be low. O'Donel Browne[2] states that a blood pressure below 60 millimetres of mercury with no improvement as a result of treatment is an indication for Cæsarean section.

The uterus loses the power of contracting and relaxing and becomes constantly tense and hard. The tension gives rise to continuous pain. The uterus also develops areas of acute tenderness. Internal hæmorrhage will further distend the impotent organ, as was shown in Case II. The degenerate state of the muscle, combined with distension, is the condition that causes labour to cease, and as distension continues it becomes impossible for labour to commence again. This assessment of the power of contraction is the most important point in the determination of all treatment. Vaginal examination, by revealing the undilated condition of the cervix and that little obliteration of the cervical canal is occurring, confirms that labour is not advancing. The bimanual examination of the uterus gives a characteristic tense sensation. Aleck Bourne[3] states that the presence of blood in the vagina is of good prognosis and is indicative of some uterine power.

In each case under observation the syndrome became progressively worse, and it was felt that, with further conservative treatment, the patient almost certainly would have died. The expulsive contractions of the uterus ceased, giving place to a state of firm tension with pain and tenderness; the uterus passively distended with blood, the pulse rate rose and the volume diminished. Pulse rates above 120 beats per minute were taken to indicate the danger zone from blood loss. These were the main indications for Cæsarean section. De Lee,[4] Johnstone,[5] Eden and Holland,[6] Francis Brown,[7] in fact most writers, state that conservative methods give the best results in accidental hæmorrhage; but they agree that Cæsarean section has a definite place in those severe cases in which there has been a poor response to conservative methods. In an attempt to compare the mortality figures in treatment by conservative methods and by Cæsarean section the absence of any uniform standard of severity makes an opinion difficult to form. At the Women's Hospital, Crown Street, during two years, 70 patients with accidental hæmorrhage were treated. The six cases reported were amongst these. Case V was the only maternal death. In the remaining 64 cases, in which treatment was by conservative measures, no case attained a severity equal to the six reported cases. Of the infants 32 either were stillborn or died after a few days. If the presence of fœtal heart sounds is an established fact, Cæsarean section offers the child the surest way of being delivered alive. Francis Brown[8] states that if more than one-third of the placenta separates the child will be asphyxiated. Prematurity is another big factor in the high infant mortality in these deliveries. Kellogg,[9] of the Boston Lying-In Hospital, quoted the maternal mortality following Cæsarean section in this condition as 22% and in a later and larger series 15%. F. C. Irving[10] gives a series of 303 cases, 200 of external hæmorrhage and 103 concealed. In 170 of the external type delivery was effected *per vias naturales*, with no maternal death; in 30 of the external type delivery was by Cæsarean section, with a maternal mortality rate of 3·3%. Of the patients with concealed hæmorrhage, 34 were treated conservatively and there was a mortality rate of 2·9%; 14·5% of 69 treated by Cæsarean section died. These figures show that Cæsarean section is reserved for the most severe cases, in which a greater mortality is expected.

The next question that arises is: "Should hysterectomy follow the Cæsarean section (Porro) or should a total hysterectomy be performed *ab initio*?" O'Donel Browne,[2] of Dublin, states that the Porro operation should never be performed, as not only is it unnecessary, but it adds greatly to the shock. Dougal[11] favours hysterectomy if the uterus fails to contract. These are examples of the opposite schools of teaching.

It is difficult to see how a much-damaged uterus can be dealt with other than by hysterectomy if it continues to bleed after the delivery of the child.

In the uteri under consideration the small dark areas of disintegration with hæmorrhage were much more numerous in Cases I, II and III. The number of these areas appears to bear a direct relationship to the power of contraction. After the injection of pituitrin and "Ergometrin" into the deltoid and the application of a hot sponge to the uterus there was no contraction in Cases I, II and III, while in Cases IV, V and VI there was enough contraction to control the hæmorrhage into the cavity of the uterus. Marshall Allan[12] states that injection of pituitrin into the uterine muscle is the best test for contraction power. Kellogg[9] states that disintegration of the muscle is no indication for hysterectomy, but bleeding after long observation with the abdomen open is the indication. Most authorities agree that if the uterus does not contract after the injection of pituitrin either into the deltoid or into the uterus and stimulation with hot pads, it is safer to perform hysterectomy. This needs a great deal of courage, as the patient can be *in extremis* at this point in the operation. To do an abdominal hysterectomy without delivery of the child by the Cæsarean wound is the treatment suggested by Berkeley, Fairbairn and White[13] in their text-book. They state that this is ideal treatment in a severe case of concealed accidental hæmorrhage with a dead fœtus. This hysterectomy has the advantage of speed; but a large experience of these cases would be necessary to choose with certainty between the uteri that would and would not contract after the delivery of the child.

The preparation of the patient, when operation has been determined, is a most important point. It is suggested that blood transfusion should be performed before and after the operation or else commenced at the beginning of the operation and continued as a drip transfusion when the patient returns to the ward. In the latter method it is important not to give too great a volume and overload the heart. It is possible that transfusion before operation may have saved the patient's life in Case XV. Intravenous injection of gum-saline solution can be life saving while a donor is being obtained; but in these severe hæmorrhages blood transfusion must be ultimately given.

No vaginal examinations or manipulations should be done in the home, because they are very dangerous. First, sepsis is always introduced, no matter how careful the examiner may be. Secondly, hæmorrhage of such severity as to be uncontrollable in the surroundings may be caused. The patient should be moved from her home at the earliest possible stage to a hospital, where blood transfusion and operation, if necessary, can be carried out with ease and without delay. In the early conservative treatment, such measures as vaginal examination, rupture of the membranes, plugging of the vagina with cotton-wool and other manœuvres should be done with every aseptic precaution. The introduction of organisms by the conservative treatment greatly increases the risk of sepsis if it becomes necessary to perform Cæsarean section.

Summary of Treatment.

A summary of the treatment adopted in these severe accidental hæmorrhages is as follows:

1. Vaginal examination should never be done in the home and should be done only with strict asepsis.
2. Cæsarean section in the most severe cases should be undertaken only after preliminary conservative treatment.
3. Hysterectomy should be performed only after stimulation of the disorganized uterus has failed to produce a contraction.
4. Repeated blood transfusion is necessary.

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THE VALUE OF WARD AND RUDD'S CULTURAL TEST FOR THE DIFFERENTIATION OF GROUP *A* HÆMOLYTIC STREPTOCOCCI.

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In June of this year, Ward and Rudd[1] described a cultural test which, they stated, differentiated with very few exceptions the potentially pathogenic group *A* strains of hæmolytic streptococci from strains belonging to the other serological groups. This test consisted in growing the strains in two specially prepared media: (*a*) serum broth, (*b*) serum peptone agar. The types of growth observed after twenty-four hours' incubation were interpreted in the following ways: (i) a feathery, semi-transparent colony in serum peptone agar medium, with or without opalescence in serum broth, suggests group *A* streptococci; (ii) a compact, opaque colony in serum peptone agar, with opalescence in serum broth, suggests group *A* streptococci; (iii) a compact, opaque colony in

serum peptone agar, without opalescence in serum broth, suggests that the streptococci do not belong to group *A*.

The feathery, semi-transparent colonies were spherical, elongated, or shaped like a Medusa head, and sometimes contained a nucleus of denser growth. Some of the opaque compact colonies were star-shaped. The only exceptions reported were two group *A* strains which, when first isolated, failed to behave in what these authors regarded as the typical manner. On subsequent cultivation both strains threw off typical variants.

Ward and Rudd made the following statement:

> There is good correlation between the information afforded by this biological test and that afforded by Lancefield's serological test.

They stated that the former method was more suitable for use in a routine laboratory. As they pointed out, the precipitin test takes time; and therefore it may be inconvenient to carry out in a busy laboratory, especially if the routine workers have to prepare their own group sera. Without doubt, many hospital laboratory workers would wish to adopt the test suggested by these authors if it regularly enabled group *A* strains to be distinguished from those of the other serological groups.

It is the purpose of this paper to report the results of the application of Ward and Rudd's cultural test to hæmolytic streptococci isolated in Melbourne.

The Material Studied.

One hundred and eleven strains from human sources were examined. Eighty-four of these were included in the series of 113 strains described by me in a previous paper;[2] the remaining 27 were isolated between April 1, 1938, and August 31, 1938. All these strains produced soluble hæmolysin when tested by the usual methods. On the surface of horse blood agar plates (either on ordinary blood agar plates or on the special plates of Ward and Rudd), the extent of the hæmolytic zones produced by different strains varied. Some of the group *B* strains and a few of the group *A* strains showed very narrow zones; but with all such strains the amount of hæmolysis was equal to, or greater than, that produced by Lancefield's group *B* strain (090 *R*). Thus there is no doubt that all of these strains would have been classed as hæmolytic streptococci by Lancefield.

Throughout their article, Ward and Rudd use the term "hæmolytic streptococci" without any modification, except that the strains were from human sources. There is, therefore, no reason why the testing of the 111 strains referred to above should not afford reliable information as to the validity of the conclusion of these authors, which was as follows:

> If hæmolytic streptococci from human sources are grown in two media, (*a*) serum broth, and (*b*) serum peptone with just sufficient agar to suspend the developing colonies, the potentially pathogenic group *A* (Lancefield) strains, with very few exceptions, can be differentiated by their growth characteristics from hæmolytic streptococci belonging to the other serological groups.

On the basis of the precipitin test, carried out either according to Lancefield's original technique[3] or according to Fuller's formamide method,[4] the 111 strains investigated by me were classified as follows:

Group *A*	83
Group *B*	12
Group *C*	7
Group *G*	9

Results.

Of the 83 group *A* strains, 81 behaved in one or other of the two ways described by Ward and Rudd as typical of this group. Thirty-two of them grew flocculently with pronounced opalescence in serum broth and as compact colonies in serum peptone agar; forty-nine grew diffusely or flocculently with or without opalescence in serum broth and produced feathery, semi-transparent colonies in serum peptone agar; the remaining two group *A* strains grew flocculently without opalescence in serum broth and gave compact colonies with down-growths in serum peptone agar.

In an investigation of strains recently isolated from human sources, Ward and Rudd encountered only two group *A* strains which behaved in an atypical manner; but the total number of strains studied was not stated. In my experience, two of 83 group *A* strains behaved atypically.

Of the 28 strains not belonging to group *A*, only 12 behaved in the fashion described by Ward and Rudd; that is to say, only 12 grew flocculently without opalescence in serum broth and as compact opaque colonies in serum peptone agar. Twelve of the remaining 16 strains not belonging to group *A* behaved in the same way as many of the strains belonging to Lancefield's group *A*; they grew flocculently in serum broth without opalescence and gave feathery, semi-transparent colonies in serum peptone agar. The other four strains gave doubtful results. They grew flocculently without opalescence in serum broth. In three instances the colonies in serum peptone agar were not so large or so transparent as the feathery colonies produced by group *A* strains, nor were they typical of the opaque compact form. The fourth gave compact colonies with down growths.

The detailed behaviour of the 28 strains not belonging to group *A* is given in Table I.

The compact, opaque colonies produced in serum peptone agar by seven of the group *G* strains differed in appearance from the compact colonies of group *A* strains. Although certainly opaque, they were larger than the group *A* colonies and slightly fluffy at the periphery.

Strains not of group *A*, giving feathery, semi-transparent colonies, were encountered both among those strains which were tested immediately after isolation and among those which had been kept on artificial culture media for periods varying from one month to two years.

TABLE I.

Detailed Behaviour of 28 Strains of Hæmolytic Streptococci not Belonging to Group A.

Strains.	Appearance in Serum Broth.	Appearance in Serum Peptone Agar.
Group B.	Flocculent growth without opalescence.	
9		Feathery, semi-transparent colony.
2		Small feathery colony, but opaque.
1		Compact opaque colony.
Group C.		
2		Feathery, semi-transparent colony.
1		Compact, opaque colony with feathery down-growth.
4		Compact, opaque colony.
Group G.		
1		Feathery, semi-transparent colony.
1		Feathery colony, slightly opaque.
7		Compact opaque colony with slightly fluffy surface.

These results may be summarized as follows:

According to the precipitin test, there were 83 group *A* strains, and 28 not belonging to group *A*; but, according to the test of Ward and Rudd, 93 belonged to group *A*, 12 did not, and with six strains the results were in doubt.

Thus, with the strains at my disposal, all of which were obtained from routine specimens, the test described by Ward and Rudd did not prove a satisfactory means of differentiating group *A* hæmolytic streptococci.

The method of preparation and inoculation of the special media was given in considerable detail by these authors. I have meticulously observed their instructions.[1]

It is therefore unlikely that my results were due to unsatisfactory conditions for growth. Rather it would appear that some hæmolytic streptococci not belonging to group *A* do not differ from group *A* strains in their capacity to produce feathery, semi-transparent colonies in serum peptone agar.

Conclusion.

The testing of 111 strains of hæmolytic streptococci isolated in Melbourne from human sources showed that the biological test suggested by Ward and Rudd could not be used to replace the precipitin test for the differentiation of group *A* hæmolytic streptococci.

According to the latter test, 83 strains belonged to group *A* and 28 to group *B*, *C* or *G*; while, according to the cultural test of Ward and Rudd, 93 were classed as group *A*, 12 as not belonging to group *A*, and in six instances the result was in doubt.

[1] The neopeptone used by me was "Difco" brand, and the powdered agar either "Misuza" or "B.D.H." The 28 strains not belonging to group *A*, and 20 of those belonging to group *A*, were tested with batches of media made up from each brand of agar. All the 111 strains were tested with at least two batches of media prepared with serum from different horses. The results obtained with the different batches of media were consistent.

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PULSION DIVERTICULUM OF THE PHARYNX, WITH REPORT OF A CASE.

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PULSION diverticulum of the pharynx has for many years been referred to as œsophageal or pharyngo-œsophageal, when in actual fact the diverticulum arises from the lower part of the pharynx. It occurs five times more commonly in men than in women, and is found between the ages of forty and eighty years, the most common age being sixty years.

Their occurrence is determined by the peculiarity of structure of the inferior constrictor muscle of the pharynx. This muscle is divided into two distinct portions, an upper portion (the thyreopharyngeus) arising from the oblique line of the thyreoid cartilage, and a lower portion (the cricopharyngeus) arising from the inferior cornu of the thyreoid cartilage, from the lateral surface of the cricoid cartilage, and from a tendinous arch convex backwards attached to these two points of origin.

In the normal resting state the cricopharyngeus is tonically contracted, acting as a sphincter to the upper end of the œsophagus, whilst the thyreopharyngeus is in a condition of partial relaxation. In the act of deglutition there is a sharp rise in pressure in the hypopharynx before the cricopharyngeus muscle relaxes. The unyielding nature of the anterior wall (the cricoid cartilage) throws the resulting pressure against the posterior wall at the junction between the thyreopharyngeus muscle and the cricopharyngeus muscle. Any gap or weakness of union between these two portions of the inferior constrictor muscle will accordingly predispose to the herniation of pharyngeal mucous membrane through the gap. It is probable that with increasing age potential weakness in the muscle junction becomes more manifest, particularly if associated with some degree of non-coordination of the lower segment of the muscle.

The chief symptoms of this condition are the following.

1. Dysphagia is the most common and the most important symptom. It varies greatly in severity, and at least in the early stages consists in discomfort in swallowing rather than actual difficulty in getting the food down. It is due to the distended pouch compressing the lumen of the œsophagus.

2. Regurgitation, not true vomiting, is usually prominent. This symptom was well shown in the case reported below, in which unchanged particles of food eaten the night before were brought back by the patient when he bent down to wash his face in the morning.

3. Noisy deglutition is very common, and often forces the patient to eat alone.

4. Hoarseness occurs, and is attributed to pressure by the sac on the inferior branch of the recurrent laryngeal nerve.

5. A gurgling sensation can very commonly be detected in the neck by the fingers when the patient is instructed to swallow.

6. A swelling in the neck may occasionally be felt. This will be found only when the sac is very large, and is an uncommon finding.

Treatment.

It is around the method of treatment that most controversy centres today. Many surgeons recommend a two-stage procedure, whereby the sac is first freed and stitched to the muscles in such a way as to give it dependent drainage, and at the second operation (performed ten to fourteen days later) the wound is reopened and the sac removed. The advocates of the two-stage method claim that in this way the dreaded complication of mediastinitis is avoided. This operation is recommended by McEvers,[1] who quotes Halstead, Murphy, Judd, Lahey and others as strongly in favour of a two-stage procedure.

Moynihan,[2] in a criticism of McEvers's article, makes a strong plea for a one-stage operation. This is supported by Shallow,[3] whose method was largely followed in the case cited below. Shallow was unable to find any reports in recent years of a high death rate from mediastinitis following one-stage operations. He quotes a series of 76 patients operated on in one stage, with only two deaths, one from pneumonia and one from uræmia. In addition, Torek[4] gives details of sixty cases with only one death. These results would suggest that the risk of mediastinitis has been somewhat over-estimated, and that eminently satisfactory results may be obtained by the single-stage method, particularly if the operation is performed in conjunction with œsophagoscopy. By this means the sac can be cleared of its septic contents and thoroughly swabbed out with antiseptic before it is opened. In addition, the sac can be readily transilluminated should any difficulty arise in locating it.

The actual technique of operation may well be described by a typical case.

Report of Case.

H.J., a male, aged seventy years, was first seen on March 4, 1938. He stated that he brought back particles of food which he had swallowed some hours previously, and the act of swallowing was difficult and was associated with gurgling. This trouble had started twenty years previously. At that time he noticed that when he bent down to wash his face in the morning he would commence to cough and would cough up some food that he had eaten the night before. Later he found that swallowing became more and more difficult, and during the last four years he had had to exercise great care in eating, taking only very small mouthfuls of food and washing them down with drinks of tea. Attacks of violent coughing would give him temporary relief from his symptoms.

On physical examination no abnormality was to be found. The patient showed no evidence of starvation, as described in some of these cases, and had lost no weight. In view of the long typical history, a provisional diagnosis of pharyngeal diverticulum was made, and this was confirmed radiologically (see Figure I). It will be seen that this is a relatively large sac with a wide mouth, and in the X ray photograph it appears to lie exactly in the middle line.

After careful attention to the mouth and teeth, operation was performed on May 24, 1938. As the vast majority of pharyngeal sacs project to the left, it was decided to make the approach from this side. Under intratracheal ether anæsthesia an incision was made along the anterior border of the left sterno-mastoid muscle, from the level of the hyoid bone to one inch above the sternum, passing through skin, platysma and deep fascia. The anterior belly of the omo-hyoid muscle was ligatured and divided, the vessels in the carotid sheath were retracted outwards and the thyreoid gland and trachea were drawn in the opposite direction. (The route of approach is shown in Figure II.) Immediately the sac was obvious, projecting to the left and rising and falling with respiration. At this stage an œsophagoscope was passed into the sac, the contents were thoroughly aspirated and the sac and lower part of the pharynx were well swabbed out with a 1% solution of "Mercurochrome". The neck of the sac was found to be unusually large, being of the same calibre as the sac itself (about one inch). The œsophagoscope was then withdrawn from the sac and passed into the œsophagus. Then the sac was dissected free, the neck was transfixed and ligatured like a hernial sac, and the sac was amputated. The stump was cauterized with pure phenol and invaginated by a figure-of-eight suture. The hernial orifice in the muscle was closed by suture of the cricopharyngeus and thyreopharyngeus portions of the inferior constrictor muscle together with interrupted stitches. The œsophagoscope was removed and the wound was closed, a small glove drain being left in the lower end.

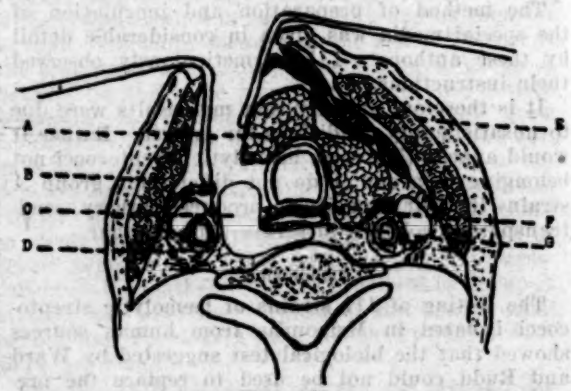

FIGURE II.

Anatomical relations of a pharyngeal diverticulum, showing route of surgical approach. *A*: thyreoid gland; *B*: sterno-mastoid muscle; *C*: diverticulum; *D*: carotid sheath; *E*: pretracheal muscles; *F*: recurrent laryngeal nerve; *G*: œsophagus.

The patient was returned to bed, and the administration of saline solution *per rectum*, by the continuous drip method, was commenced. Twenty-four hours later the glove drain was removed and the patient readily swallowed a Ryle's tube, through which glucose in saline solution was given at frequent intervals. This tube was removed at the patient's request on the second day after operation, and thereafter small quantities of fluids were given by mouth. The wound healed by first intention and the patient was discharged completely symptom-free on the tenth day after operation.

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Reports of Cases.

PATHOLOGICAL REPORTS FROM THE CHILDREN'S HOSPITAL, MELBOURNE.

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XV. CARCINOMA OF THE COLON IN A CHILD.

THE occurrence of carcinoma of the colon at any time after the completion of the third decade of life occasions no surprise, and during the fifth decade and later it is depressingly commonplace. It is by no means uncommon between the ages of twenty and thirty years; but few would expect to meet it in a child who, at the time this manifestation of malignant disease originated, had not completed her tenth year. She was, in fact, aged nine years and nine months when the disease entered its clinical phase, although it can be well understood that it was not immediately recognized. When, in June, 1933, I received the specimen of which Figure XXIII is a photograph, and identified it as an adenocarcinoma of the sigmoid, I thought that a clinical record had been established. However, consultation of the relevant literature, as will be shown, proved that the little girl who provided this specimen was by no means the youngest subject in whom a carcinoma of the colon had been recorded. The effect of a perusal of the literature was to make me feel inclined to echo the querulous complaint of Ecclesiastes: "Is there anything whereof it may be said, See, this is new?"

The clinical notes concerning this patient are very lengthy, but it is desirable to recount the leading features. The surgical problems involved were capably handled by Dr. Charles Osborn, but it is outside my province and beyond my capacity to discuss them.

Clinical History.

The patient, at the time of her admission to the care of Dr. Charles Osborn at the Children's Hospital, on June 16, 1933, was aged nine years and eleven months. Two months prior to that date she became ill with abdominal pain and diarrhœa, the bowels acting four or five times a day and the motions containing blood and mucus. Treatment for colitis was instituted by her private medical attendant, symptoms were alleviated, and the girl appeared to improve in health until May 20, when she again suffered abdominal pain, accompanied by nausea and vomiting and recurrence of diarrhœa. Blood and mucus reappeared in the motions. During the week immediately preceding the child's admission to the Children's Hospital the diarrhœa had ceased, but intermittent abdominal pain persisted.

On her arrival in hospital the child was febrile and looked ill. Physical examination disclosed a smooth, rounded, tender, non-mobile mass situated in the mid-line in the hypogastrium. The clinical notes describe the mass as about the size of a coconut, dull to percussion and tender to palpation. The area of tenderness embraced the whole of the hypogastric region. Rectal examination occasioned no pain or tenderness apart from that caused by stretching of the sphincter; but a certain fullness in the mid-line, apparently continuous with the hypogastric mass, was noted.

Dr. Osborn decided that surgical intervention was imperative, and operated within twenty-four hours of the child's admission. By means of an incision made a little to the left of the mid-line of the hypogastrium, he exposed an indurated mass, enveloped in turgid and œdematous omentum, lightly adherent to the abdominal wall and to the contiguous loops of small intestine. No difficulty was experienced in mobilizing the mass, the location of which was determined as the sigmoid colon, and in the doing of this a small abscess was ruptured. Dr. Osborn proceeded with resection, by the method of Mikulicz, establishing the colostomy inherent in this technique. At this stage he formed the opinion that tuberculosis, in the indurated, hyperplastic and tumour-like form in which it frequently manifests itself in the colon, was the most probable solution of the pathology of the condition.

The child made a good immediate recovery; but convalescence was protracted. Her vicissitudes included an attack of nasal diphtheria, but the principal factor making for a prolonged stay in hospital was delay in the closure of the colostomy. On October 7, 1933, Dr. Osborn presented this girl at a meeting of the Melbourne Pædiatric Society, at which time she was very well and the colostomy seemed to be closing. At the end of December a certain amount of fæcal matter was still being discharged through the artificial anus. The girl was then sent home for a few weeks, pending operative closure of the colostomy. This was effected by Dr. Osborn on March 3, 1934, and the patient, to all appearances very well, was finally discharged on March 27, 1934.

Three years later, on April 26, 1937, she reappeared at the Children's Hospital with a complaint of headache and vomiting, recurring in attacks which were becoming more frequent and severe. A detailed neurological examination, carried out by Dr. E. Graeme Robertson, yielded as the salient findings slight blurring of the upper edges of the optic disks, fine irregular nystagmus, generalized hypotonia, and an extensor response in the plantar reflex on the right side. On May 2, six days after her readmission to hospital, and a few weeks short of four years after the original operation, the girl collapsed suddenly and died almost instantaneously.

Post Mortem Examination.

An objection to *post mortem* examination on the part of the patient's relatives was partially overcome by Dr. Howard Williams, who prevailed on them to consent to an examination of the brain. It was not possible, therefore, to determine the extent and distribution of recurrent malignant growth, if any, within the abdomen; but in the brain was found a tumour protruding from the ventral surface of the right lobe of the cerebellum (Figure XXIV). The presenting portion was roughly circular in shape and approximately four centimetres in diameter. The growth had obviously undergone a spontaneous hæmorrhage and was thereby rendered very friable. No neoplasm could be found in the cerebrum.

The nature of the colonic tumour provides very little scope for discussion, in that it presented no contentious features. Who ran might read in the microscopic section the simple histology of an advanced columnar-celled adenocarcinoma. Colloid change was not a prominent feature, but fields in which it had been initiated could be found with a little search.

Reference to Figure XXIII will show that the growth was of considerable superficial extent and was of the type which fungates into the lumen of the bowel, as distinguished from the scirrhous type, which in its natural evolution results in the production of an annular stricture. The neoplasm had replaced the normal mucous membrane of the bowel over its whole area for a length of ten centimetres, and had effected reduction of the lumen to the extent shown in the illustration. There was much superficial ulceration of the tumour tissue, and concomitant infection appeared to be the only factor in the enlargement of several lymphatic glands excised with the tumour. Microscopic sections of the glands showed an intense and acute inflammatory process of a suppurative character. It

was to suppuration in a lymphatic gland that the small abscess evacuated at the operation was most probably attributable, as there was nothing approaching deep ulceration or perforation in the bowel.

Examination of a microscopic section of the tumour in the cerebellum confirmed the presumption that it was a metastasis from the original carcinoma. It was distinctly an epithelial growth, and the acinous arrangement of the primary tumour was reproduced more or less faithfully (Figure XXV). Occasional fields showed acini filled with colloid material, but, as compared with the primary colonic tumour, there was not much stroma.

Discussion.

Had it been permissible, it would have been very interesting to investigate conditions within the abdomen *post mortem*. Text-book teaching is that in this type of carcinoma of the large bowel glandular involvement and extension of the growth beyond the intestine occur relatively slowly. The girl had displayed no symptoms indicative of spread of malignant disease in the abdominal viscera; but this fact scarcely justifies an assumption that there had been no recurrence.

Of an interest at least equal to that of the question of recurrence would have been the examination of the colonic mucous membrane for possible predisposing polyposis. The most frequent site of intestinal polyposis is the rectum, and at intervals in past years at the Childrens' Hospital rectal polypi have found their way to the pathological department in routine fashion; but I have never known one exhibit malignant properties in microscopic section. Recurrence of such polypi and subsequent malignant metaplasia might well have occurred in one or more of the children so affected; but as such an event would be delayed beyond the age at which patients pass from observation at the Children's Hospital its development would be very difficult to trace.

The term "polyposis of the colon" should be restricted to connote adenomatous hyperplasia of the intestinal mucous membrane only, and not extended to embrace those polypoid tumours of the intestine which bear the histological stamp of fibroma, myoma, lipoma or angioma. Erdman and Morris[1] divide intestinal polyposis into adolescent and adult types. The peculiar features of the adolescent or congenital disseminated type are the countless numbers in which the polypi occur, their wide distribution in the intestinal mucous membrane, and the strong tendency of this form to occur in several members of the same family. In the adult type, secondary to traumatic and chronic inflammatory lesions, for example, amœbiasis, the polypi occur in limited numbers over a relatively restricted area of the mucous membrane.

It should be emphasized that the polyposis in both congenital disseminated and adult types shows a pronounced predilection for the large intestine, and the relevance of the introduction of intestinal polyposis into the present discussion lies in the fact that it manifests a striking tendency towards the development of malignant adenoma and adenocarcinoma. The incidence of malignant change in polyposis of the colon has been assessed by G. Hardy[2] as more than 40%. The same writer quotes Lockhart Mummery[3] as stating that "almost all cases of multiple polypi of the colon eventually become malignant".

Whether underlying polyposis was present in this child will never be known. The occurrence of carcinoma of the colon at such an early age suggests the possibility of some such special tissue predisposition, and an inspection of the colonic mucous membrane generally would have been an important indication had a routine autopsy been obtained.

As already indicated, the case of this child is not without precedent. Ewing,[4] indeed, in discussing carcinoma of the colon, speaks of the "considerable number of cases occurring in children". With all due deference to such a high authority, it is perhaps permissible to state that the fact that only one instance has occurred in the twenty-five years for which pathological records exist at the Children's Hospital, Melbourne, caused me to read this statement with some surprise and to speculate on the degree of elasticity inherent in the term "considerable". I am free to confess, however, that after consulting the literature I did not feel quite so much disposed to criticize Ewing's phraseology.

In the section dealing with carcinoma of the colon in Allbutt and Rolleston's "System of Medicine"[5] a reference is made to a specimen of colloid carcinoma of the sigmoid which had recently been sent to the pathological department of the London Hospital, as having come from a boy, aged ten years; and it is stated that other cases of colloid cancer of the colon have been put on record (Kanthack and Furnivall, W. A. Garrard). From the same source I learned that Nothnagel quotes other instances at seventeen, twelve, eleven, three and a half and even three years of age.

It was interesting to find in the Medical School Library of the University of Melbourne, the volume of the *Transactions of the Pathological Society of London* for the year 1897, containing the case report by Kanthack and Furnivall,[6] relating to a carcinoma of the large intestine in a boy, aged seventeen years. Another case report is that of W. A. Garrard,[7] also published forty-one years ago, the medium being *The Quarterly Medical Journal*. This journal, not to be confused with *The Quarterly Journal of Medicine*, served the northern counties of England for eleven years only; the complete file is to be found in the library of the Victorian Branch of the British Medical Association. In the number for April, 1897, I read Garrard's report of a case of carcinoma of the colon occurring in a child. The patient was a boy, aged twelve years, in whom a carcinoma of the sigmoid loop of the colon occasioned intestinal obstruction. Pathological verification that the primary growth was a colloid, columnar-celled carcinoma arising in the sigmoid flexure, was supplied by Dr. J. Targett, one of the curators of the Royal College of Surgeons.

Lockhart Mummery[3] mentions Dr. Porter Parkinson as having reported the case of a boy, aged five years, in whom had arisen a carcinoma of the sigmoid flexure. The *Quarterly Cumulative Index Medicus* records that H. J. Warthen[8] recently reported an instance of cancer of the colon in childhood in the *Virginia Medical Monthly*, a journal to which I have not been able to obtain access.

In a discussion of carcinoma of the large bowel Theodore S. Raiford[9] has published an analysis of the age incidence of all subjects of cancer of the rectum admitted to the Johns Hopkins Hospital over a period of forty-two years. In a total of 511 there were 319 examples of carcinoma of the rectum, and 192 of carcinoma of the colon. Of the 192 colonic cancers, none occurred in the first decade of life, two in the second.

C. H. Phifer[10] collected from the literature 49 cases of cancer of the rectum or sigmoid in persons under twenty years of age. In this series there was only one in the first ten years of life, if an example occurring in a monster be excepted. This author appears to have searched the Continental literature thoroughly, and quotes Weinlechner as having determined, among 5,279 cases of carcinoma of the colon, 18 in children up to fourteen years of age. Phifer also comments on the fact that in the young, as in the adult, carcinoma of the lower part of the digestive tract exhibits a decided predilection for the sigmoid.

Thus it would appear that, after all, Ewing was right in his use of the expression "considerable" as applied to the number of cases of carcinoma of the colon which have been recorded in childhood. How little I knew when I thought that the instance which came under my observation would establish a record! Verily, to quote the preacher again, "there is no new thing under the sun".

Acknowledgement.

I am obliged to Dr. Charles Osborn for his consent to my suggestion that I should utilize the clinical record and pathological specimens relating to his exceptionally interesting patient for one more essay in the present series.

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PROLAPSE OF THE IRIS TREATED WITH TRICHLORACETIC ACID.

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Clinical Record.

ON July 2, 1938, Mrs. C., aged thirty-nine years, was referred to me by Dr. H. O. Rock, who had just seen the patient for the first time. Her history was that on June 13 she was combing her hair when the comb slipped out of a knot in her hair and struck her on the upper eyelid of her left eye. Her eye felt a little sore, but did not greatly distress her. However, as some discomfort persisted, she eventually consulted Dr. Rock, who sent her to me.

On examination no mark was seen on the eyelid, but the pupil was pear-shaped. On her looking downwards, a large prolapse of the iris was seen at the limbus at the twelve o'clock position. The prolapsed iris was entangled in and matted up with the overlying conjunctiva, so that an iridectomy would have been by no means a simple matter. Indeed, it probably would have been most unwise, in view of the disturbance it would have created in the eye, which was quiet and white, except for a tinge of redness around the actual prolapse. There were no keratic precipitates.

Treatment by applications of trichloracetic acid in saturated solution was decided upon, and the acid was applied on July 4, 5, 7, 9, 12 and 15. On the last date it was obvious that the prolapse was rapidly flattening out, but at the same time a plentiful crop of keratic precipitates was observed. This changed my attitude from one of satisfaction to one of anxiety; and visions of sympathetic ophthalmitis came into my mental view. Atropine, which had previously been instilled only after each application of acid, was now used three times a day, along with hot foments and large doses of sodium salicylate. A blood count revealed no abnormality; and as time went on the keratic precipitates slowly lessened in number, while a constant watch with the slit lamp revealed no aqueous flare in the unaffected eye.

Eventually all keratic precipitates disappeared. The patient was left with a quiet eye, a good large pupil and visual acuity of $^{6}/_{6}$ with a +0·75 cylinder (vertical axis).

Discussion.

The features of special interest in this case are: first, the quietness of the eye three weeks after a perforating wound of the eyeball; secondly, the successful flattening of the prolapse with trichloracetic acid; and thirdly, the appearance of keratic precipitates subsequent to a series of applications of the acid.

Reviews.

THE SICK INFANT.

THE latest edition of Porter and Carter's "Management of the Sick Infant and Child"[1] contains many new features of interest.

The book is divided into three sections: The first includes discussions of such disturbances as vomiting, diarrhœa, constipation, malnutrition, hæmorrhage, pain and tenderness, convulsions, fever, cough and prematurity. The second section deals with the treatment of the system diseases. The last is devoted to special methods of treatment, therapeutic procedures and clinical laboratory examinations. The value of this third section is enhanced by its excellent detailed descriptions of the technique of intravenous infusion and of blood transfusion; the preparation of fluids for intravenous therapy; the subcutaneous and intraperitoneal administration of fluids; lumbar, cisternal and ventricular puncture; gastric lavage and gavage; collection of sputum; and methods of examination and treatment. This section concludes with chapters devoted to food formulæ and recipes, drugs and poisoning.

There are a certain number of statements in this book which do not conform to accepted pædiatric teaching, particularly the recommendation of the intraperitoneal route as a satisfactory one for blood transfusion and the statement that a child suffering from dysentery "should receive a total of 200 cubic centimetres or more" of dysentery antiserum. Despite these aberrations this book is a useful and practical aid for both the pædiatrician and the general practitioner.

DEAFNESS.

THE authors of "The Handicap of Deafness",[2] Irene R. Ewing and Alex. W. G. Ewing, are well known in the little world of educators of the deaf, and have been responsible for valuable pioneer work in the modern development of apparatus for measuring, conserving and educating residues of hearing. The preface of the book shows how greatly the special work of aiding a sorely afflicted section of the community continues to depend on the interest and generosity of great-hearted citizens, and states that the Department of Education of the Deaf in the University of Manchester, possibly the most valuable and progressive institution of this kind in the United Kingdom, was the result of the beneficence of the late Sir James E. Jones, of Rochdale, Lancashire.

The book is based on years of carefully gathered laboratory, clinical and classroom experience. It covers a wide range of experimental effort. The case records given are of particular interest. We could not, however, accept the arguments and conclusions of the authors without further and far more extensive investigations. We prefer to regard the book as a record of faithful and scientific work, which will clarify the issues of the problem it deals with, rather than as a final solution of the problem.

We cannot agree with the statements of the authors that there is no such thing as a clear-cut border-line between the deaf and dumb and that there is a large section of the population of every age that suffers from defective hearing. While these views may be true to some extent from the physical and sensory, and perhaps from the more psychological standpoints, they are certainly less true from the educational and pedagogical standpoints, for in educational practice the distinction between those whose hearing experience has been sufficient to give a working basis

[1] "Management of the Sick Infant and Child", by L. Porter, B.S., M.D., M.R.C.S., L.R.C.P., and W. E. Carter, M.D.; Fifth Edition, revised; 1938. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay (Surgical) Proprietary Limited. Royal 8vo, pp. 874, with illustrations. Price: 63s. net.

[2] "The Handicap of Deafness", by I. R. Ewing, M.Sc., and A. W. Ewing, M.A., Ph.D.; 1938. London: Longmans, Green and Company. Demy 8vo, pp. 337, with illustrations. Price: 12s. 6d. net.

of naturally acquired spoken language and those who have been deprived of that experience and advantage, is definite and vital. It is undoubtedly important that we should, as the authors have tried to do, establish the best possible physical and sensory conditions for making use of the degree and nature of residual hearing, and bear in mind constantly the possible future progress of that hearing. But it is equally important that we should at the same time work from the other angle and find a better, if not the best, means of presenting speech and language to those for whom their acquisition remains a slow and laborious classroom process.

It has become recognized as a danger that the development of the modern hearing-aid method of educating those deaf children (the minority) who are capable of benefiting therefrom, and to whom it is an inestimable boon, may affect adversely the interests of those children (the majority) for whom the method presents no present promise.

The statement that 80% of the deaf have some hearing is probably true. If and when the hearing that is retained can be used educationally, it should be so used. But enthusiasm should not oust common sense, and if a child's hearing will not help him to acquire language, it is educationally valueless to him.

There are much interest and some inspiration in the book, but it is too technical in form and specialized in purpose to realize the publishers' hope that it will be of great value to the general public. Its main appeal will be to the teacher of the deaf and perhaps to the general practitioner, who is often called on to give preliminary advice to deaf patients. It contains little that will be new to the otologist.

RADIUM APPLICATORS.

IN the foreword to the excellent monograph by D. G. Walker, "The Construction of Vulcanite Applicators for Applying Radium to Lesions of the Buccal Cavity, Lips, Orbit and Antrum",[1] we read that "the treatment of malignant disease of the mouth has undergone a marked change in recent years. Radium has superseded and supplemented surgery in the majority of cases". It is by meticulous care in technique, such as is emphasized in this publication, and by accurate estimation of dosage that radium therapy has attained its present position in this field. Radium work is a specialty which should be undertaken only by those who are adequately trained and who work in centres where the necessary organization and means are available.

An early chapter of the book is devoted to physical considerations, and in it the advantages of the surface application of radium as opposed to interstitial implantation are admirably summed up. It is interesting to note that physical considerations have led to the wider use of radium moulds in mouth lesions and the extension of their use to the antrum and other sites.

The protection of the surrounding healthy tissues, an essential point in mouth mould construction, is obtained either by the incorporation of lead in the applicator or the displacement of these tissues to a greater distance from the radium by the addition of more vulcanite. Mr. Walker appears to favour lead protection in general. It has the disadvantage, however, of adding considerably to the weight of the applicator, and it is probable that the use of distance for protection is quite sufficient in practice.

The greater part of the book deals with the technique of construction of various types of applicator. Quite a large section is devoted to methods of fixation of the applicators, and many ingenious devices are described. It is stated that improved results have followed the use of these methods, and it is asserted that the time taken in construction is of little consequence when the technique used is one of routine in a well-organized department.

The book is rich in excellent photographs, so that each step in technique can be followed with ease. It contains much of value both to the radium therapist and to the dentist who is asked to assist in the application of radium.

[1] "The Construction of Vulcanite Applicators for Applying Radium to Lesions of the Buccal Cavity, Lips, Orbit and Antrum", by D. G. Walker, M.A., M.Dent.Sc., B.Ch., with a foreword by W. W. James, O.B.E., F.R.C.S., L.D.S.; 1938. London: John Murray, for the Middlesex Hospital Press. Demy 8vo, pp. 70, with 23 plates. Price: 7s. 6d. net.

DISEASES OF THE CHEST.

DR. JAMES MAXWELL, author of "Introduction to Diseases of the Chest", is to be congratulated on having produced an excellent text-book intended primarily for students.[1] The general arrangement of the book, the clearness and lucidity of the text, and the absence of unnecessary and confusing discussion will make an instant appeal to the student who wishes to master the essentials of respiratory disease. The practitioner also will find in it much to interest and stimulate him. He will appreciate a book that is easy to read, and will welcome it as a reliable guide to modern practice in this field of medical endeavour.

The opening chapters are concerned with a discussion of the symptoms of respiratory disease and with physical examination of the chest. Then follows an account of special investigations. The last and largest section of the book deals with the actual diseases of the respiratory tract. Each section is handled splendidly. A few points deserve special mention. The author rightly insists on the value of physical examination. This is timely advice in days when there is a tendency to allow X ray examination to displace ordinary physical methods. But he also very rightly emphasizes the value, indeed the necessity, of special investigations, without which in many cases accurate diagnosis is impossible. The chapter on the radiology of the chest is a particularly instructive one. Dr. Maxwell recommends screen examination as well as skiagraphy. The text is illustrated by a good series of radiographs.

The section on diseases of the respiratory tract commences with a discussion of lesions of the upper air passages, and the author describes many common conditions in the nose and its accessory sinuses, which have an important connexion with disease in the lower part of the respiratory tract, but which are often overlooked.

When a text-book is of such obvious merit, any criticism seems out of place. In speaking of the diagnosis of pulmonary tuberculosis the author does a service by insisting that the absence of physical signs is no excuse for the omission of special investigations. But not everyone will agree with the statement that tuberculin tests are of no practical value in diagnosis. And if the advice is followed that a positive diagnosis should be made only after the demonstration of tubercle bacilli, then many cases will be missed in the earlier and more curable stages. But these minor points do not detract from the value of a book that should be in the possession of every student interested in the study of diseases of the respiratory tract.

[1] "Introduction to Diseases of the Chest", by J. Maxwell, M.D., F.R.C.P.; 1938. London: Hodder and Stoughton Limited. Demy 8vo, pp. 340, with illustrations. Price: 12s. 6d. net.

A TEXT-BOOK OF SURGERY.

STILL another text-book of surgery, by Dr. C. F. W. Illingworth, of Edinburgh fame, comes to our notice.[2] Concise and easily read, short and well spaced, this book will prove a boon to student and practitioner. The only notable fault is the complete absence of any description of operative surgery. The orthopædic and fracture sections are particularly well done. There is no mention of lumbar puncture in the chapter on head injuries; this portion is somewhat disappointing.

The illustrations are numerous and well selected. Altogether this book gives a good up-to-date synopsis of the fundamentals of the majority of surgical conditions.

[2] "A Short Textbook of Surgery", by C. F. W. Illingworth, M.D., F.R.C.S.; 1938. London: J. and A. Churchill Limited. Medium 8vo, pp. 710, with 8 plates and 179 text figures. Price: 21s.

The Medical Journal of Australia

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NATIONAL HEALTH INSURANCE IN AUSTRALIA.

NATIONAL health insurance in Australia was anticipated for many years before the Bill was brought before Parliament. It had been discussed on many occasions by politicians and by the profession. Occasional threats of its introduction were made and were met by mild protests or gentle cries of alarm. In the course of time medical men became accustomed to the bogy, and despite the warnings of their leaders, ignored it. Consequently, when the Bill at last came, the announcement of its provisions struck like a thunderbolt. The profession reeled for a moment, then leapt into action. In the early heat the features most discussed were those that appeared most prominently and thrust themselves most violently before the observer. Almost every argument was concerned with money. Little was said, except by a few thoughtful people, of the possible effect of panel practice on professional morals and efficiency, or of the possible value or danger of national health insurance to the community, or of the necessity or desirability of any form of national health insurance in Australia. Even the scope of service received scant mention. But the fear of reduced incomes was not the only reason for this attitude. The Bill was not truly a health bill; it was a financial measure, brought in by the Commonwealth Treasurer, not the Minister for Health, and, so we believe, was designed primarily to relieve the Government of its large expenditure on pensions. Money was its main consideration.

As a result of the profession's protestations a Royal Commission was appointed to inquire into certain aspects of national health insurance. Partly as a result of the type of the profession's protestations and partly because the Act was sponsored by a financial rather than a health expert, the terms of reference of this Commission are concerned solely with money matters—the remuneration of medical practitioners under the Act. It seems that to a financial expert and indeed to some medical men, all things appear mensurable in pounds, shillings and pence.

Now that the more excitable members of the profession have become sobered and the less excitable have had time to think, many are asking why the Commission is not presented with broader views on the nationally important features of health insurance. The answer is in the terms of reference. Still, it is not for us to say whether any particular evidence is or is not admissible. If a man has something of importance to say, he should seek an opportunity of saying it while there is still time. Many things must be said and many things done before national insurance will be acceptable to the profession in Australia. For the proper working of any such scheme the goodwill of the profession must be secured.

It has been officially announced that the health provisions of the Act will not come into operation until May, 1939. This will give time for the formulation of a scheme that will be acceptable to the nation and the profession alike. Masses of data are available now that were unobtainable before the drafting of the Bill. If the Federal Executive of the British Medical Association were to meet the Government now, its members would be provided with sufficient information to enable them to discuss national health insurance on an equal footing with the Government's experts. Formerly they had no

reply to the experts' arguments. They stated the case for the profession, but were unable to substantiate their claims. They had to accept as correct the figures and statements presented to them; for they themselves had none. And it was on these figures and statements that the Bill was based. Now is the time for the Federal Council to collaborate with the Commonwealth Department of Health, the health departments of the various States, and other informed bodies, in the evolution of a scheme of health insurance that will be a true health insurance, designed for the welfare of the poorer people, and including in its scope not merely a few services for the wage earner, but good general services for the unemployed and the unemployable and for the breadwinner and his dependants.

It may reasonably be doubted whether national health insurance is required in Australia. If we must have it, let it be in such a form that it will give us cause for pride and will be a credit to our nation.

Current Comment.

THE TOXICOLOGY OF ERGOTAMINE TARTRATE.

ON November 6, 1937, we made reference in these columns to a communication by S. Perslow and L. Bloch in *The Journal of the American Medical Association* on the subject of gangrene caused by ergotamine tartrate, which is extensively employed in the treatment of migraine. T. J. C. von Storch has now given the results of an extensive study of the complications following the use of this substance.[1] It is prepared under the trade name of "Gynergen". Von Storch states that, since the isolation of ergotamine tartrate by Stoll in 1918, 42 serious *sequelæ* of its use have been reported, but not one has been described in connexion with the treatment of migrainous headache. In 1921 K. Spiro maintained that this preparation had not been proved to produce gangrene in man. He reiterated the statement in 1929 after a more extensive study. He insisted that ergot produced a dry form of gangrene, whereas the variety attributed to ergotamine was moist. H. Guggisberg collected from the literature ten cases of gangrene in which this preparation had been employed during the puerperium. He considered that puerperal infections predisposed to the occurrence of gangrene and often caused it without the aid of ergot. Accordingly he doubted whether ergotamine tartrate was responsible. H. Saenger held that this drug was only an accessory malefactor. Von Storch states that, of the 42 recorded "accidents" following the use of this drug, 22 happened in obstetric patients, 11 in sufferers from hyperthyreoidism, and eight in persons with miscellaneous disorders. Various manifestations, ranging from anginal attacks to "impending gangrene", developed in 20 patients, gangrene in 21, and death in eight. In 18 cases the fault lay in overdosage, and in 16 sepsis was a contributory and possibly the causative factor. A few patients had coexisting hepatic or renal disease. Gross overdosage was responsible for one of the eight fatalities, coexisting sepsis and obliterative vascular disease for two, coexistent coronary disease for one, and ergotism and hepatic cirrhosis for one. For three no cause could be ascertained, but autopsy did not reveal evidence of ergotism. A study of the literature shows that sepsis, obliterative vascular disease and associated cardio-vascular disorders, apart from overdosage, have been important factors in producing the ill effects attributed to ergotamine tartrate. Von Storch, however, admits that, although in the majority of cases there are significant predisposing factors, there remain some instances in which ergotamine tartrate itself must be held responsible.

Von Storch conservatively estimates that over the past twenty years the percentage of accidents must have been considerably less than 0·01. He states that the manifestations of ergotism resulting from the use of ergotamine tartrate have not been clearly defined. These manifestations have always been determined by the symptoms that follow overingestion of whole ergot. But it must not be forgotten that whole ergot has constituents, such as histamine, that might change or mask the phenomena due to ergotamine tartrate alone. Whole ergot may cause gangrene or convulsions or a combination of both. Convulsions, however, do not invariably occur in "convulsive ergotism", and it has therefore been suggested that "neurogenic ergotism" would be a preferable designation. In this type the outstanding symptoms are fatigue, heaviness of head and limbs, giddiness, insomnia, excitement, delirium, dementia, mania and paræsthesiæ of hands and feet, generally but not always bilateral. Occasionally pseudo-tabetic signs are evident. Hemiplegia or paraplegia may occur. Histological examination may reveal degeneration of the optic nerve and of the dorsal and lateral columns of the spinal cord. The changes in the cord resemble those of *tabes dorsalis* or, more frequently, the type of degeneration noted in deficiency diseases. It has been suggested that "convulsive ergotism" is actually determined by a coexisting deficiency of vitamin *A*. As many of the cases occur in famine districts, it is more than possible that a state of hypovitaminosis exists. Von Storch points out that, while children are con-

[1] *The Journal of the American Medical Association*, July 23, 1938.

sidered to be more prone to ergotism than adults, there have been no reports of ergotism in children following the use of ergotamine tartrate. No untoward reactions have followed its use in children for migraine. When the unfavourable manifestations which sometimes follow the use of ergotamine tartrate are compared with those due to whole ergot, it will be noticed that those caused by ergotamine tartrate are similar to the milder manifestations of convulsive ergotism. This suggests that the factor causing neurogenic or convulsive ergotism may be ergotamine. But other derivatives of ergot may induce similar manifestations. Further, ergotamine tartrate may produce either type of ergotism in animals or man. Von Storch holds that it may induce ergotism indistinguishable from that caused by whole ergot. Experimental gangrene and accidental gangrene in man due to ergotamine tartrate are pathologically similar.

The vascular changes are of special importance. In general there is an arterial vasospasm primarily affecting the arterioles and smaller arteries. Accompanying this spastic condition are varying degrees of intimal œdema and hyperplasia, hyaline degeneration, thickening of the arterial walls, lymphocytic infiltration and frequent thromboses. The capillaries have been found dilated. Venous vasoconstriction is present in varying degrees, but is not so marked as the arterial. Although Spiro held that ergotamine tartrate was not a vasoconstrictor, and Straub believed that gangrene due to it was the result of vasomotor paralysis, there is much recent evidence to the contrary. Von Storch maintains that uncomplicated gangrene due to ergot is dry in character until sepsis supervenes.

ALARMING SYMPTOMS DUE TO GLYCERYL TRINITRATE.

THE interest shown of recent years in pain of cardiac origin has resulted in an increased use of certain vasodilator drugs, particularly trinitrin or glyceryl trinitrate. Formerly numbers of vasodilators were popularly employed in the attempted treatment of hypertension, but, perhaps fortunately, most of these are quite inert or are merely transitory in their effect. Now their use is practically restricted to the relief of pain or related discomfort in cases of coronary ischæmia, the two preparations found most reliable being amyl nitrite and glyceryl trinitrate. The former is subject to the objection that its administration is by inhalation. It is pungent and rapid in action, and therefore even dosage is difficult or impossible to attain, especially when the drug is self-administered. Glyceryl trinitrate is more simply given as a tablet, though care should be taken that the tablet is chewed or allowed to dissolve in the mouth to ensure adequate absorption.

The assumption that glyceryl trinitrate is perfectly harmless is general, but Harold C. Lueth and T. G. Hanks have pointed out that severe reactions may occur after its use.[1] They report that nausea, vomiting and a definite degree of collapse occurred in a number of their patients, and although no residual ill effects were noticed, the immediate results were in some cases quite alarming. In most of these cases the patient had a fairly high grade of arterial hypertension, and it is well recognized that many hypertensive subjects have a very labile circulatory system and a sensitive carotid sinus reflex. In order to investigate the possibilities of severe reactions following the administration of trinitrin, Lueth and Hanks tested fifty patients suffering from essential hypertension who were attending an out-patient dispensary. To ensure a uniform method and dosage they used a graduated capillary pipette and delivered accurately measured amounts of a standardized alcoholic solution of glyceryl trinitrate under the tongue of the patient. Estimations of blood pressure were made at frequent intervals and careful note was taken of the patient's subjective symptoms. Peripheral vascular changes were estimated by various experimental methods, such as the use of a finger plethysmograph, the injection of histamine and the study of capillaries in the nail bed. Although all the observations were made under standard conditions, in no case could any prediction be made as to the likelihood of the occurrence of severe reactions. Perhaps this result was to be expected, for it does not follow that undue lability in the capillary circulation in the limbs indicates a parallel state in the whole vascular system. The symptoms of collapse occurred in nine patients out of fifty, or 18%, a number which seems surprisingly high. These symptoms included nausea, vomiting, syncope, collapse and involuntary sphincter action, and were observed in one or two patients after a dose as low as 0·2 milligramme (one three-hundredth of a grain). They were naturally more severe if the patient was sitting up than if he was recumbent. Complete recovery took place within an hour in every case.

Some of the authorities quoted by Lueth and Hanks have described death following the administration of this drug, but the quantities taken have been relatively large, such as one-tenth of a grain. Apparently the lethal dose for man is not known. It seems very unlikely that any really dangerous symptoms will occur after a therapeutic dose, but it is well to point out that this drug, widely prescribed and generally considered harmless, may cause alarming manifestations. Probably susceptible patients have some vascular idiosyncrasy. Nevertheless we must remember that all useful drugs are potentially toxic, and one of the first requisites for their safe administration is as accurate a knowledge as possible of the uncomfortable or even dangerous reactions they may set up.

[1] *Archives of Internal Medicine*, July, 1938.

Abstracts from Current Medical Literature.

RADIOLOGY.

Hæmoptysis and Röntgen Examinations.

G. W. HOLMES (*Radiology*, August, 1938) states that there is a small group of cases of hæmoptysis of tuberculous origin in which the physical and X ray examinations of the chest reveal no abnormality, although the patient may have as a presenting symptom a profuse pulmonary hæmorrhage, and in some cases tubercle bacilli are found in the sputum. While it is difficult to prove beyond a doubt the actual source of disease in cases of this type, a considerable percentage are due to ulcerations in the bronchi. In some at least calcified glands can be shown or bronchoscopic examination reveals a definite erosion. The author states that older physicians will remember the term "lung stone", about which we hear little today. He believes that these calcified masses found in the sputum were undoubtedly, in some instances, glands which had ulcerated through into a bronchus and were then expectorated. Such an accident is usually followed by blood-spitting, possibly by severe hæmorrhage, and occasionally by the appearance of tubercle bacilli in the sputum. X ray examination in such a case frequently fails to reveal evidence of a pathological process in the lung substance, but may show other calcified masses.

Peritendinitis Calcarea.

CARL SÄNDSTROM (*The American Journal of Roentgenology*, July, 1938) states that *peritendinitis calcarea* is a definite disease entity. The characteristic feature of the condition is calcification in the soft tissues, found on Röntgen examination and located in tendons and tendinous tissue, ligaments and articular capsules, and in their surrounding connective tissue. The clinical picture and course of the peritendinitis may be described as having the acute, chronic and latent forms. The shadows seen in acute cases are often thin and of cloudy character, and ill defined from the surrounding tissue. In cases of longer standing the shadows are often dense, well set off from the surroundings, and homogeneous. In other cases they appear as small granules or drops, partly confluent with large irregular shadows. No structure is seen in the shadows; sometimes there is an indication of a stratified arrangement. The calcifications obviously are then localized in certain preformed spaces of the soft tissue. Often the shadows are exceedingly small, appearing as conglomerations of tiny points. In such cases it may be impossible to make a diagnosis from the Röntgenogram alone. The calcifications are localized partly to tendons and tendinous capsular and ligamentous tissue of coarse trabecular structure and containing few cell nuclei, partly to undifferentiated cellular connective tissue surrounding joints and tendons. In no case were the calcifications found in a bursa; in one case calcification was found possibly in the wall of a bursa. In some, probably of longer standing, the calcifications were inside small cavities lined with endothelium and located within or outside a tendon. In all cases the chief treatment was Röntgen irradiation, and excellent results followed. Prior to Röntgen treatment many patients were given various kinds of heat therapy without any benefit.

Resolving Lobar Pneumonia in Adults.

SAMUEL COHEN (*Radiology*, August, 1938), in discussing the X ray appearance of resolving pneumonia, states that the criterion for the radiographic diagnosis of the onset of resolution is a decrease in the extent or intensity of the opacity characteristic of the stage of hepatization. The first site of resolution is variable. It may manifest itself in the peripheral or hilar zones or in the centre of the dense shadow. A diffuse haziness is frequently an early finding, which may disappear rather uniformly or rapidly, or more commonly becomes broken up into smaller areas of lighter infiltration, which are patchy in distribution. Röntgenograms of the thorax, repeated at intervals of several days, often reveal complete disappearance at some point, while in others infiltration may persist. This corresponds to the pathological evolution of the process. Many times linear streaks are seen extending from the hilum in all directions; they cross one another and also intersect larger areas of infiltration, giving the appearance of a reticulum. It is difficult to say whether these lines are due predominantly to vascular or lymphatic dilatation. In a lung field so disorganized the presence of annular shadows is not infrequent. These may be due simply to a ring-like configuration of markings or actually to distended air sacs. The infiltration disappears, the lacunar shadow or shadows become thinned out and clear, and the pulmonary markings are the last to take on their normal appearance. In the differentiation between pneumococcal pneumonia and pulmonary tuberculosis, one of the most important points hinges on the time interval for resolution of the pulmonary process. Resolution in pneumonia may first occur at the time of the crisis, but may also antedate or follow it. While there is considerable variation, disappearance of the infiltrative and ring shadows usually takes place in seven to fourteen days after the crisis in uncomplicated cases. Another significant point is the absence of residue of the pulmonary infection on Röntgen examination as resolution is completed. Pulmonary tuberculosis, on the other hand, resolves much more slowly, over periods of weeks and months, and during the process of healing a residue in the form of strands or nodules of fibrosis or calcium deposits is seen in the Röntgenograms. The difference between the two infections in these respects is the difference fundamentally between a destructive and a non-destructive process in the lung. Ordinarily, pneumococcal lobar pneumonia is really a superficial infection of the lung, producing no necrosis of alveolar or bronchial walls or of interstitial tissue. This accounts for the rapid and complete return to normal.

Urography in Pregnancy.

E. ROHAN WILLIAMS (*The British Journal of Radiology*, May, 1938) states that physiological pelvi-ureteric dilatation and adynamia in normal pregnancy are caused by obstruction from the enlarging uterus, hypertrophy of the lower part of the ureter, and the effect of hormones. Obstruction from the enlarging uterus seems such an obvious cause that the tendency is to accept it uncritically. That this is not the sole factor is shown in those cases in which great dilatation occurs very early in pregnancy, long before the uterus could exert any pressure upon the ureters, and by the fact that a few women go to term without the occurrence of any dilatation. *Post mortem* studies on pregnant women without urinary infection have shown that a concentric hypertrophy occurs in the lower zone of the ureter. The constriction that this causes is further increased by a hypertrophy of the ureteric sheath in the form of an encircling ring. These changes would certainly account for some degree of obstructive dilatation, but hardly for the adynamia. The presence of hormones probably plays a part, since there is something in the blood of pregnant women that prevents the uterus from emptying itself until full term. While this uterine inhibition is present so also is there a relative adynamia of the pelvi-ureteric musculature. As both the ureters and uterus have similar embryological origins, similar smooth muscle and a similar nerve supply, the author considers it reasonable to apply this inhibition theory to account in part for the physiological changes, especially the adynamia. The radiological diagnosis of *placenta prævia* should be extremely accurate with the aid of cystography. A preliminary study before cystography should not be omitted. The bladder is catheterized and emptied and 60 to 90 cubic centimetres of sodium iodide solution are instilled; it is important that the bladder should not be distended. Radiographs are then taken in the antero-posterior and

the two oblique planes. Centring must be accurate at a point in the midline at the upper symphysis level. Moderate downward pressure should be applied to the uterine fundus during exposure. A radiograph with the patient in the erect position should also be made when feasible. The diagnosis of *placenta prævia* depends upon an analysis of the translucent space between the contrast margin of the fundus of the bladder and the fœtal skull outline. With our present knowledge this method is reliable only in vertex presentations. The total thickness of the space should not exceed 6·0 to 8·0 millimetres in normal cases. If this soft-tissue gap is appreciably widened, it is almost certain that some abnormal structure is present in the uterine segment, the walls of which are very thin. This abnormal structure must be either a blood clot or a *placenta prævia*. The actual spacing of the gap will indicate the type of placental implantation on the lower segment, whether central or whether lateral or marginal.

PHYSICAL THERAPY.

Intestinal Injuries after Radium and Röntgen Treatment of Carcinoma of the Cervix.

JAMES A. CORSCADEN, HAIG H. KASABACH AND MAURICE LÆNZ (*The American Journal of Roentgenology*, June, 1938) state that after an increase in radium and Röntgen ray dosage for carcinoma of the cervix a group of injuries of an ulcerative type occurred. These were limited to the mucosa of the intestine. In the cases reported, the symptoms, physical signs and findings at operation or autopsy presented pictures of varying degrees of intestinal injury, from the mildest type, causing a simple proctitis, through various stages of destruction to gross necrosis, terminating in perforation or cicatricial repair with varying degrees of stenosis or atrophy. The radiation injuries were eliminated without reduction either of the total milligramme-hours or X ray dosage administered. This was accomplished in several ways. The number of milligrammes used in the utero-vaginal applications was reduced from 175 to 70, and the duration of the application was increased from 40 to 100 hours. The daily Röntgen dose was reduced from 300 or 400 *r* to 100 or 200 *r*, and the size of the field was reduced from 20 by 20 centimetres or 15 by 20 centimetres to 10 by 15 centimetres or less. The duration of the course of treatment was increased from between twenty-one and thirty days to forty or more days. After this change in technique the incidence of intestinal injuries, which had been at the rate of 8·7%, dropped to nil. The cases are reported together with the dosage factors for the following purposes: (1) to point out to those who may observe the occurrence of intestinal obstruction or ulceration of the rectum or sigmoid in patients who have been treated for carcinoma of the uterus, that the condition may be the result of radiation injury to the intestine and may not be caused by carcinomatous involvement; (ii) to draw attention to the possibility that diarrhœa, abdominal distress and nausea occurring during treatment may be symptoms of a definite injury to the intestinal mucosa and not of a general radiation sickness; (iii) to warn the therapists who, by increasing their radiation dosage to a level necessary for efficient cancer therapy, run the risk of causing similar injuries; and particularly (iv) to emphasize the importance of the time factor in dosage.

Importance of Statement of Dose in Radiotherapy.

J. R. NUTTALL (*Radiology*, May, 1938) deplores the confusion of expression in the statements of dosage factors for both radium and X ray therapy published by various clinics. The aim of the radiotherapist is to deliver a dose of irradiation that will be lethal to the tumour, and to give it in a manner as little harmful as possible to the surrounding normal tissues. It would appear reasonable, therefore, for him to think, speak and write in terms of the dosage it is necessary for him to give to the tumour he is treating. Exact knowledge of tumour radiosensitivity is directly dependent upon knowledge of the response of tumours of similar structure to known doses of irradiation. Hence the importance of a statement of "tumour dose" as the salient item on the routine treatment record sheets. There are many technical difficulties in the realization of a universal standard description of dosage, quite apart from the difficulties directly related to differing personal opinions. A reasonable assessment of tumour dose can, however, be stated in any treatment carried out in conformity with modern physical conceptions of radiotherapy; and it is better to make a reasonable or approximate assessment in cases in which the physical difficulties are great than not even to attempt to state the dose used. A plea is therefore made for the practice of stating tumour dose as the salient statement in the description of radiotherapeutic technique, on the grounds that dosage is the basic criterion of any treatment, that it is essential for accurate knowledge of radiosensitivity, that it would simplify the teaching of radiotherapy, and that it would greatly aid correlation of the results obtained in various clinics.

Radium Therapy of Carcinoma of the Cervix Uteri.

H. H. BOWING AND R. E. FRICKE (*The American Journal of Roentgenology*, July, 1938) survey the results of treatment of 1,491 patients with carcinoma of the *cervix uteri* at the Mayo Clinic during the years 1915 to 1929. Radium therapy has been used at this clinic in the treatment of this disease for the last twenty-two years. Classification of the lesions into stages was made according to the recommendations of the League of Nations, and a histological examination of tissue removed was carried out in every case. The majority of lesions, as would be expected, were epitheliomata. Radium therapy was given by the intensive broken-dose method. It is interesting to note that the treatments, with very few exceptions, were given with the patient in the knee-chest position. As a rule, all patients received supplementary deep X ray therapy. In this large series of cases, although the great majority of patients (91%) were in an advanced stage of the disease, 26·8% of the entire number lived for five or more years in apparently good health after treatment; also 69·2% of patients with lesions in Stage I were well at the end of five years, and 60·2% of those with borderline lesions were well at the end of five years. That there is little risk from the treatment is shown by the fact that the hospital death rate for the entire series was only 1%, the mortality occurring in the group with advanced lesions.

Low-Voltage, Near-Distance X Ray Therapy.

J. F. BROMLEY (*The British Journal of Radiology*, May, 1938) discusses the general principles of low-voltage, near-distance X ray therapy and the clinical experience acquired during two years' practical work with the tube associated with the name of Professor Chaoul, of Berlin. He prefers the original term "contact therapy", by which this method of treatment was first described. He points out that any tumour the surface of which cannot be brought into actual contact with the applicator, is unsuitable for the method. However, in tumours of the tonsil, palate or alveolus sufficient access may often be provided by removal of the teeth, and in rectal growths access may be provided by removal of the sacrum. In the treatment of basal-cell carcinoma of the canthus region or of the nose a dose of 4,000 *r* over about twelve days is used by the author. He advocates a dose of at least 6,000 *r* for other carcinomatous tumours. He has found the method very valuable in the treatment of recurrences in the skin after radical amputation of the breast and of extensive fungating epitheliomata of the dorsum of the hand. He has also studied the effects of this form of X ray therapy on the histology of tissues to which it is applied, by selecting two immediately adjacent skin nodules in an advanced case of breast cancer and excising them under local anæsthesia after one has been irradiated. He is convinced that contact therapy is a most valuable addition to the armamentarium of the radiotherapist.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on August 25, 1938, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. G. M. BARBON, the President-Elect, in the chair.

Muscular Dystrophies and Atrophies Occurring in Childhood.

DR. A. R. HAMILTON read a paper entitled "Muscular Dystrophies and Atrophies Occurring in Childhood" (see page 887).

DR. R. J. TAYLOR read a paper entitled "Muscular Atrophies and Dystrophies in Childhood" (see page 889).

DR. E. H. M. STEPHEN said that one consoling thought was that most of the conditions under discussion were rarities. It was impossible to give any kind of estimate of how frequently they occurred. A comparatively large number of cases of pseudo-hypertrophic muscular dystrophy were encountered. There now appeared to be some hope for these patients if they were treated with ephedrine or with the new drug mecholin. Dr. Stephen said that he would like to hear more about the type of patient likely to be benefited. Erb's juvenile type of progressive muscular atrophy, the scapulo-humeral type, was not common. As for the facio-scapulo-humeral type, he had not seen a patient whose facial muscles were involved. He had been interested by the description of the physiological changes in muscle and by the possible advances in knowledge made by the estimation of the creatine and creatinine excretion in a number of cases comparatively recently. Dr. Stephen had seen one patient suffering from pseudo-hypertrophic muscular atrophy, in whom Professor Lambie had been interested. The patient had been given theelin for a period of six or seven months without improvement. This case was the only one in which Professor Lambie and Dr. Stephen had used theelin. One child suffering from the same pathological condition had had an unusual distribution of weakness of muscles, the abdominal muscles being affected. Dr. Stephen had previously believed that the only muscle in this region likely to be affected was the *latissimus dorsi*, but in this case the internal and external oblique muscles were apparently affected. A large number of sausage-shaped tumours developed. These proved to be scybalous fæcal masses, which the child had insufficient muscle power to expel. Dr. Stephen had not seen weakness of the abdominal muscles in pseudo-hypertrophic muscular atrophy mentioned in any text-book. Another patient with advanced pseudo-hypertrophic muscular atrophy had been referred to the orthopædic surgeons and admitted to Canonbury Convalescent Home. Some time later Dr. Stephen was delighted to see the child advancing to meet him. He had been enabled to get about by means of very ingenious calipers. This was a remarkable achievement and made the child extremely happy. Such children, when they reached the period of their disease which meant inability to get about, were in an extraordinarily unhappy state. Dr. Stephen said that he was glad to know that such patients, in their very unfortunate condition, could get this relief.

DR. LORIMER DODS asked Dr. Taylor how soon after treatment with ephedrine and potassium chloride he had noted clinical improvement in these cases. Dr. Dods had recently treated a child, aged five years, apparently suffering from a pseudo-hypertrophic muscular dystrophy. The child was given 0·03 gramme (half a grain) of ephedrine three times a day and three grammes of potassium chloride per day. After less than six weeks of such treatment this child had shown a most surprising degree of improvement. The rapidity of this child's recovery had suggested the possibility that the original diagnosis might have been incorrect. Dr. Dods was glad to hear Dr. Taylor emphasize the futility of creatine and creatinine excretion tests as a method of investigating these dystrophies. Disturbances of creatine and creatinine metabolism were not responsible for the disease, but were effects of it. Dr. Dods pointed out that although the average case of muscular dystrophy was not associated with any endocrine disturbance, there were rare cases in which both disturbances were present. Dr. Taylor had mentioned the syndrome of *dystrophia myotonica*, which suggested a combination of an endocrinological disturbance (progeria) and myopathy. Dr. Dods had recently been asked to see an extremely wasted boy, aged twelve years, whose history and clinical examination suggested the possibility of Simmond's disease associated with a distal type of myopathy. Dr. Dods felt that careful investigation of mixed cases of this type might shed some light on the cause of the muscular dystrophies.

DR. T. Y. NELSON said that he had been interested by Dr. Hamilton's reference to congenital deformities. He had tried to find out something about the pathology of their causation, but the matter was far from being settled. Dr. Nelson referred first to the "tibial kyphosis" of Middleton. He said that this condition was included as a muscular dystrophy; but he questioned whether the muscular element was the principal deformity or whether it was due to a combination of factors. Professor Inglis preferred to group it with other congenital malformations, such as absence of the tibia, birth fracture, pseudarthrosis *et cetera*. All these could be grouped as deformities due to defects in the germ plasm, and regarded as different grades of one pathological condition. The weak point in Middleton's classification was that this condition had been found when no abnormality in the muscle was present. It had been suggested that it was due to venous obstruction, but it was hard to imagine how the venous obstruction came about. It had been suggested that congenital torticollis was due to venous obstruction causing an ischæmic paralysis similar to that present in Volkmann's contracture, a condition seen most commonly in the muscles of the forearm when their blood supply was obstructed by pressure in the antecubital fossa. Conditions in the sterno-mastoid muscle did not seem to be quite comparable with those in Volkmann's contracture. The question of venous stasis had been taken almost to its extreme limits by Denis Browne, who had tried to explain arthrogryposis by the same means. This condition was frequently associated with hydramnios. It was common in lambs, and shepherds said that they could tell when such a lamb was going to be born by the size of the ewe's belly. Browne's argument was that an increase in the amniotic fluid meant compression of the fœtus, with venous stasis and consequent ischæmia. One difficulty in its acceptance lay in the fact that although hydramnios frequently accompanied arthrogryposis, it did not invariably do so. Dr. Nelson said that he had mentioned these various theories only to show that the pathology seemed to be not clearly worked out. No one could speak with certainty in these matters, but a consideration of such theories added to the interest of this study.

Dr. Taylor, in reply, said that he had found the work in preparation of his paper most fascinating. He was grateful to Dr. John Eccles, of Sydney Hospital, whose work on the physiology of nerve-endings in muscle was well known. In reply to Dr. Edgar Stephen, who had asked about ephedrine and potassium chloride, Dr. Taylor said that both ephedrine and potassium chloride, as well as eserine, had acted well in the case of the girl whose illness he had described. These substances, like prostigmin and guanidine, all acted on the motor nerve-endings, making the impulses to muscle fibres stronger and more frequent. Dr. Taylor considered that eserine was easier to give, as it could be given by mouth, whereas prostigmin had to be given by hypodermic injection. He had great hopes of guanidine, which had been found very effective in *myasthenia gravis*. Guanidine given by mouth was completely absorbed by the stomach. He had had no personal experience of replacement therapy, as he had not tried cholinesterase. Dr. Stephen had never seen facial involvement in Erb's type of progressive muscular

atrophy. Dr. Taylor said that he had under treatment at that time a girl who had had double ptosis and an entirely expressionless face. She had improved greatly since she had been having ephedrine. Her expression was better, but the ptosis had not diminished. Another patient was a boy, who had advanced pseudo-hypertrophic muscular paralysis and who had had to be wheeled about in a chair. After treatment for a month or two with ephedrine and potassium chloride he could get himself about on his arms and knees, which was a slight improvement. The boy had since been lost sight of. Dr. Dods had asked what time usually elapsed before improvement was noted. Dr. Taylor said that in the case of the girl he had mentioned improvement occurred in a fortnight.

Dr. Hamilton, in reply, agreed with Dr. Nelson that the pathology had not yet been finally settled. With regard to "tibial kyphosis", Middleton had definitely considered its relation to other congenital abnormalities, but there was no muscle change in these other deformities. Middleton seemed to have satisfied most people that torticollis was due to venous obstruction.

MEETING OF THE COUNCIL OF THE VICTORIAN BRANCH.

A MEETING of the Council of the Victorian Branch of the British Medical Association was held on September 28, 1938, DR. J. NEWMAN MORRIS, Chairman of Council, in the chair.

Alien Medical Practitioners.

The Medical Secretary reported that he and the Honorary Secretary had interviewed the Minister, who appeared to be not in favour of any amendment of the *Medical Act* that might lessen the possibility of obtaining medical officers for Mallee areas, particularly Manangatang. It was resolved:

> That an *ad hoc* committee be appointed to consider the position of country areas without doctors, draw up a scheme for meeting the situation, and draft an agenda for discussion with interested authorities and bodies, that the committee consist of Professor Marshall Allan (convenor), Dr. Vernon Davies and Dr. G. Raleigh Weigall, with power to coopt, and that the Honourable the Premier be advised of the action taken.

Organization of the Profession in the Event of War.

The Chairman referred to the position in Europe and the apparent imminence of war. He stated that a committee had been formed, on which the Federal Council was represented, to consider the provision of medical services to the civilian population in the event of war. A secret report had been submitted by the committee, but he was at liberty to disclose that the report recommended the appointment of a central committee with committees in each State, on which the British Medical Association would be represented. The duty of the committee would be to allocate those doctors available, to arrange for hospital accommodation *et cetera*. A security mobilization was to take place immediately, to be followed by a general mobilization should there be a threat of invasion. A form was to be issued to all doctors in Australia, asking for disclosure of personal particulars, financial obligations, willingness and ability to serve, and in what capacity *et cetera*. Consideration would have to be given to what steps would have to be taken to conserve the interests of those called up. No complete scheme was yet in being.

Dr. V. Davies asked how far the information was confidential, and was told that he was at liberty to mention it to members of his subdivision.

Dr. Ross asked would the question of conservation of practices be regarded as urgent, as four doctors in Geelong might be called up immediately.

Dr. Norris stated that one of the security troop battalions had been deprived of its medical officer through illness and it might be necessary to replace him within forty-eight hours. He asked any members present to supply him with the name of any doctor who would be willing to enlist at once for service within Australia.

Salaries of Medical Officers of Health.

The Medical Secretary reported an interview with Dr. Merrillees, at which Dr. Merrillees had expressed the opinion that the minimum salary of £50 *per annum* for medical officers of health, as suggested by the Commission of Public Health in 1920, was too high. He referred particularly to the position at Woodend, from which Dr. Lincoln had resigned and was seeking the minimum rate. Dr. Merrillees had intimated that he was going to use all his efforts to see that the salary was not paid, and if necessary that two shires would amalgamate and employ one medical officer of health.

It was resolved:

> That a letter of protest be sent to the Commission of Public Health and that members in the Woodend district be informed that council would strongly disapprove of any member accepting an appointment which would stultify the efforts made on Dr. Lincoln's behalf.

National Health Insurance.

The following recommendations submitted by the Combined Organization and National Insurance Subcommittees were adopted without amendment:

> (*a*) That representations on behalf of the profession be made before the Royal Commission in respect of reference VI.
>
> (*b*) That the combined committees accept the principles and recommend to council that in any scheme which includes dependants: (i) The principle of loading should be applied to cover at least a section of the dependants. (ii) That for the efficient administration of a voluntary medical service to dependants a statutory body fulfilling the following requirements should be created on a State or territorial basis, such statutory body to be empowered to receive funds and use them solely to develop health services. (iii) That the development of health services should be the sole consideration of such body. (iv) That there should be adequate medical representation to ensure a realization of the required services and the harmonious cooperation of the medical profession.

The following recommendation was adopted after amendment by the addition of the word "wife" after "dependant".

> (*c*) That for the implementation of these principles the combined subcommittee recommends to council the memorandum prepared by Dr. Embelton, provided that the amount paid for each dependant be not less than that for the insured person and provided also that the figures be substantiated by reliable evidence.

It was resolved:

> That the resolutions be forwarded to the Federal authority dealing with national insurance evidence before the Royal Commission as the policy of this council and that the Federal authority be urged to make sure that Dr. Embelton gives evidence along these lines to the Royal Commission during its sittings in Victoria.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Alpins, Oscar, M.D. (Bologna), 109, Collins Street, Melbourne, C.1.

Proceedings of the Royal Commission Appointed to Inquire into Matters Pertaining to National Health Insurance.

Monday, November 14, 1938.

The sittings of the Royal Commission were resumed at 2.30 p.m. on Monday, November 14, at Hobart.

Mr. W. J. V. Windeyer and Mr. J. D. Evans announced their appearance with Mr. C. A. Weston, K.C., on behalf of the British Medical Association. It was stated that Mr. Weston would not be present at the Hobart sittings.

The Chairman: Before the business proceeds, I wish to say, on behalf of the Commission, how greatly we lament the terrible happening by which five gentlemen in the prime of life, who were rendering valuable assistance to the country, who had won our hearts by their courtesy, have come to an untimely end. We think it fitting before we resume the business of this Commission that we should pay our tribute to those whom we have lost.

Mr. Dovey: May it please the Commission: I desire on behalf of those who are appearing before this Commission, to add our tribute to the memory of those fine men who worked with us for so long and were taken from us in such tragic fashion. The friendly atmosphere in which this inquiry was being conducted was made possible to a large degree by the efficiency and charm of manner of those counsel and the gentlemen assisting them who have now been taken from us. The legal profession in Australia is the poorer for their passing, and we, members of that profession, mourn the loss of colleagues who endeared themselves to all with whom they came in contact. Once again we offer to their sorrowing relatives our deepest sympathy.

Mr. Dovey informed the Commission that he intended to call evidence on behalf of the Government of the State of Tasmania, indicating the nature and scope of the medical service provided by that Government through paid medical officers in certain areas of the State. The friendly societies, he stated, did not propose to be represented at the Hobart sittings.

Mr. Dovey further stated that he hoped to call, when the Melbourne sittings were resumed, a member of the National Insurance Commission to give evidence as to the attitude of that Commission upon a number of matters that had been referred to them for their consideration.

Mr. Dovey also informed the Commission that it appeared to him that it was undesirable to have different scopes of service for insured persons and for the dependants of insured persons, and that therefore he had suggested that a standard scope of service should be agreed upon by the friendly societies in Victoria and New South Wales. He stated that the Insurance Commission agreed with him in this matter and that arrangements had been made for representatives of the friendly societies of New South Wales and Victoria to confer and, if possible, to arrive at some form of standard scope of medical service, which, if satisfactory to the medical profession and to the Royal Commission, would, he understood, be accepted by the Insurance Commission in lieu of the scope of service outlined by Mr. Brigden in the early stages of the inquiry.

Mr. Windeyer stated that he did not contest the desirability of having the scope of service to be rendered to dependants the same as the scope of service to be rendered to insured persons.[1] Mr. Windeyer further drew attention to the difficulty in which the representatives of the British Medical Association were placed by reason of any change at this stage of the proposed scope of service.

The Chairman: The suggestion is that the scope of service which may be ultimately prescribed by the Commission for insured persons may be somewhat narrower than that which was outlined previously by Mr. Brigden, but not to any very material extent. It will be somewhat narrower; that is to say, it will be brought into line with the scope of service which has prevailed, generally speaking, in New South Wales and Victoria, as I gather, and the difference will not be very great. I do not say that it is not a matter you will not have to consider and we will not have to consider, but I do not see how it is going to very materially embarrass you in applying the evidence or inferences from the evidence as given to that somewhat narrower scope of service.

Mr. Windeyer: It may be that it will not. All I really can ask is that, at the earliest possible moment, we should have defined, and defined in a manner which we hope will be permanent . . .

The Chairman: There is no doubt that, as soon as possible, you should know what is going to be done.

Dr. John Francis Gaha, of Hobart, was called by Mr. Dovey. Dr. Gaha is a member of the Legislative Council of Tasmania and Honorary Minister for Health in that State.

Dr. Gaha gave evidence concerning the scheme of medical service recently introduced by the Government of Tasmania into certain defined areas of that State, nine in number, which are comparatively sparsely populated and in which there had previously been no, or comparatively little, medical service available.

Mr. Dovey: Will you just tell us in broad terms what is the nature and the scope of the service which has been introduced by the Government in those districts?

A.: We set out to meet the situation in the only way we thought practical in the circumstances, and we decided to put in full-time medical officers free for everybody. These doctors have certain definite duties, certain definite hours, certain prescribed holidays, certain opportunities to do refresher courses in hospitals, and there are fixed fees, but the fees are mostly in relation to after-hours. We were afraid that the services of such doctors might be abused, and we put on the after-hour fees more as a deterrent than with a desire to gain anything. The areas mentioned have very scattered populations, and the stimulus to the scheme arose out of the fact that we had observations made which indicated that there was a considerable amount of chronic ill health in these areas.

Q.: By reason of the fact that medical services had hitherto not been available?

A.: Yes, we attributed it to that, anyhow . . .

Q.: Taking it as it is at present, what is the system of remuneration of the doctor?

A.: We pay the doctor £700 a year, *plus* £50 house allowance, *plus* 6d. per mile.

Q.: For all travelling?

A.: Mr. Tudor will tell you the finer details. We intend to give the better medical officers an extra £150 at the end of three years, if they prove to be satisfactory. That is to say, we will give them £50 a year extra, so their salary really will be £750, *plus* £50, *plus* 6d. per mile, *plus* £1 1s. for night calls, *plus* 10s. 6d. for calls at their surgery, and *plus* certain special midwifery fees if the people in these areas will not go to the Bush Nursing midwifery centres provided.

Q.: You say 6d. per mile, the doctor providing his own car?

A.: Yes.

Q.: As ministerial head of the Department of Health, can you tell the Commission whether any difficulties have been experienced in getting suitable men to take these jobs?

A.: We have had plenty of men offering. If we have had any difficulty at all, it has been the difficulty of selection. Up to the present, with one exception, through ill health, we have chosen rather well. We have had a lot of experienced men offering—men with seven to eight years' experience.

Q.: Was there any proposal about furlough or holidays?

A.: We were a little too liberal in what we decided. At one time it looked as though certain professional

[1] *Vide* Term of Reference Number VI.

pressure might be brought to bear to obstruct us, and we were trying to establish the scheme by pacific means. At that time national insurance was not in the air, and I suppose the medical profession could afford to be a little more austere than it can today, and we thought that professional pressure might be brought to bear on us and an attempt made to frustrate our scheme. We had no real grounds for thinking that, but it looked as though it might possibly move in that direction. If pressure had been brought to bear, it might have made it difficult for us, and the only thing we could do in the circumstances was to try to create certain reasonable conditions to attract reasonable medical men. With that object in view, we decided to give them a month's holiday, to which I have no objection, seeing that they live in remote areas, but we also decided to give them a month's refresher course in our hospitals. I would like the Commission to know that the hospitals in Tasmania differ fundamentally from the hospitals on the mainland.

Q.: I was going to ask you to deal with that aspect very shortly?

A.: Very well. But the doctors get a month's refresher course in our hospitals and a month's holiday a year, which, in my considered opinion, is too liberal.

Dr. Mulvey: Two months a year?

A.: Yes.

Q.: And do you allow a post-graduate course as well?

A.: That is the refresher course.

Q.: Of one month per year?

A.: Yes.

Mr. Dovey: Do you propose to adhere to that?

A.: For the ones we have now we have to adhere to it. There is no written agreement, but there is an honourable understanding, and I do not intend to digress from it.

The Chairman: He is a Government officer, giving the whole of his time, at a salary from £700?

A.: Yes, *plus* £50 house allowance, *plus* 6d. per mile, *plus* £50 per year if he gives satisfactory service.

Q.: And his prospects are those of a Government officer. That is to say, if he stays on in the job, he continues to get the salary and allowance?

A.: Yes.

Q.: Is there any chance of his getting further advancement, or is he to get that as a fixed remuneration for life?

A.: At the moment he has to take it as fixed, but that will depend upon Government policy in the future, and it will depend largely upon the trend of medicine in Australia. If the trend is towards more Government control, then bigger positions will be offering, and naturally men of more experience in these services, who look like competent administrators, will get up in the service. In Tasmania we have perhaps half a dozen positions better than this, and I venture to say that if these men show a degree of competency in the administration of their particular districts they will have first chance of the other positions.

Dr. Mulvey: Is there any provision made for promotion of these appointees?

A.: Not up to the present.

The Chairman: And is there any retiring age fixed?

A.: Not by statute.

Q.: That means, I suppose, that so long as a man is competent and sticks to his work, you will not interfere with him until he shows signs of approaching age, but then what is going to happen?

A.: My answer is that we considered that matter and were of the opinion that the salary we are giving him, *plus* the allowances and the opportunity, is above the average.

Mr. Dovey: You had in mind, in regard to these remote areas, the net income in a country district?

A.: We consider that a medical practitioner in these districts, with reasonable economies, can make very nearly as much as the average medical practitioner. I am not speaking now of specialists, but of the general practitioner. It is the same class of work as he is called upon to do in these areas as a private practitioner.

The Chairman: It is the average earned by medical practitioners in similar areas; is that what you had in mind?

A.: Yes.

Q.: Have you any data upon that point?

A.: No. I have arrived at it rather by my own personal experience of practice, discussions with those with whom I am intimate, and by a good deal of questioning of medical men throughout Australia.

Evidence was then given by Dr. Gaha as to the extent and nature of hospital accommodation in Tasmania.

Dr. Gaha was cross-examined by Mr. Windeyer concerning the Government medical service. It was stated that further details of the service would be given by Mr. Tudor, an officer of the Department of Health, whom Mr. Dovey proposed to call later. In conclusion Mr. Dovey asked: I understand the local authority or Government authority exercises some discipline on the patient who unnecessarily called out a doctor?

A.: Yes. We have asked the doctors to report any abuse of privilege, and up to the present we have only had one case, and the circumstances were such that ignorance could have explained the whole thing. We intend to exercise exacting measures on anyone who abuses privileges we extend to them, and we have told the districts that.

Dr. Albert William Shugg, of Hobart, was examined by Mr. Windeyer.

Mr. Windeyer: Are you a lodge medical officer at the present time?

A.: No, I have not had any lodge patients for the past six or seven years. When I originally came to Hobart I bought a lodge practice and developed it, but later resigned from some of the lodges and ultimately from the balance.

Q.: I understand that in this State the rates paid to medical practitioners for lodge practice are not uniform?

A.: That is so; they are not uniform.

Q.: Did you have any written contract with the lodge when you were last doing practice?

A.: No.

Q.: What was your capitation fee when you ceased lodge practice?

A.: One pound per head per male lodge member in Hobart.

Q.: Did that include their dependants?

A.: Yes.

Q.: Did you have any females?

A.: I had a few. Their capitation fee was fifteen shillings.

Dr. Shugg then gave evidence as to certain discussions and movements by the profession in Tasmania to secure a higher capitation fee, which, however, had proved abortive.

Mr. Windeyer: How many lodge members did you have when you gave up lodge practice?

A.: When I finally gave up I had 600 lodge members, but previously when I resigned from half of them I had between eleven and twelve hundred.

Q.: What caused you to give it up?

A.: Firstly, I considered the capitation fee was too small and there was far too much work and too little money. Secondly, I felt that with a large lodge practice one could not do the type of work or the adequate degree of work that I thought one should do.

Q.: When you say you had twelve hundred lodge members, was that twelve hundred lodge members and their dependants in addition?

A.: Yes, their dependants in addition.

Q.: What has been your experience in hospital accommodation in regard to obtaining beds for patients in the public hospital?

A.: We rarely have difficulty at all. In the past there seems to have always been ample room for immediate and urgent cases, but for chronic cases there is now definitely a waiting list.

Q.: In regard to your experience of lodge practice, was there an income limit for the lodge members?

A.: There was supposed to be.

Q.: Did you enforce it?

A.: I did on about two or three occasions in which I knew there were glaring examples of breaches of that contract and I refused to treat the patient.

Q.: Apart from those cases, did you have other lodge patients who were outside of the income limit to your knowledge?

A.: Yes, there was quite a few scattered amongst them.

Dr. Shugg then gave confidential evidence concerning his practice and income. He estimated that as a result of the introduction of national health insurance he would lose 26% of his practice.

Mr. Windeyer: In your opinion, what will happen to such dependants of members of friendly societies as become insured persons?

A.: I have a number of lodge patients who come to me privately, and a number of their dependants come to me privately at the present moment, and most of the services that I render to those patients, both dependants and members of the lodges, are, practically speaking, included services. If they come under the national insurance scheme, they will be completely lost to me as a source of revenue.

Mr. Dovey: Why?

A.: Because, if they are under the scheme at the present moment, I can neither give them certificates nor prescriptions, and consequently, it is most unlikely that they will follow their usual procedure of coming to me for any private attention, because they can neither get their sick pay nor their prescriptions. That is one reason. There is another reason. I do not know, of course, what is going to happen ultimately, but my original assumption was that the lodge members would pay their lodge dues for their dependants and their dues for national health insurance, with the result that they would have very much less chance of coming to me with a 10s. 6d. fee than they would have in the past. That is my attitude. As far as the excluded services are concerned, they do not affect me particularly, because I do very little of them, as far as other lodge patients go.

Mr. Windeyer: You have told us that you think your practice will be affected by national health insurance in that way. Have you formed any conclusion at all as to whether you will resume contract practice under national insurance?

A.: My attitude in the past and at present is that undoubtedly I would not take on any further contract practice, either under lodge or national health insurance schemes, but that, of course, is not a final word, until one realizes fully what the terms of service and every detail connected with national insurance will be. But my present judgement, if I may say so, is that I certainly would not revert to a less efficient service, such as I consider is given to contract or panel type of work.

The Commission adjourned at 4.32 p.m. until Tuesday, November 15, 1938, at 10.30 a.m.

Tuesday, November 15, 1938.

Mr. Edward Joseph Tudor, Secretary of the Department of Public Health in Tasmania, was examined by Mr. Dovey.

Mr. Tudor gave evidence concerning the medical scheme which Dr. Gaha had previously outlined, detailing certain modifications and amendments in the scope and conditions of service under that scheme. Statistics were given showing the population, attendances, mileage fees and private fees in the various districts in which the scheme operated. Bush nursing services in Tasmania were also described.

Mr. Tudor was cross-examined by Mr. Windeyer generally in relation to the scheme and then the witness was questioned by Dr. Mulvey.

Dr. Mulvey: Comparing your contract service with that of a general practitioner, you pay him £750 a year, and the doctor who acts as his *locum* for two months, that is £120, and in addition to that you pay travelling allowances up to £180, assuming it is 5,000 miles' travelling?

A.: It would be over 5,000 in some cases.

Q.: Assuming that is so, that would make the income £1,140?

A.: Yes.

Q.: Then there is a £50 bonus paid if he is there for three years, and you also pay for his instruments?

A.: Yes.

Q.: That is roughly £1,200 a year?

A.: We estimate that each medical officer will cost the Government £1,000 a year on the present figures.

Q.: That is including *locum* services?

A.: Excluding *locum* services.

Q.: He is one of your employees, is he not?

A.: Yes.

Q.: So that as far as the income of the practitioner under the Government contract is concerned, he is really receiving about £1,200 compared with a private practitioner?

A.: I would say £1,000.

Q.: One thousand pounds for every general practitioner, but for a practitioner in the Government service in the country?

A.: It costs the Government more than £1,000, but the Government man will not receive an income in excess of £1,000.

Q.: But compared with a private practitioner it would be roughly £1,200?

A.: Yes.

The Chairman: For twelve months' work the doctor receives £700, which may grow to £800, roughly speaking. He receives a subsidy to some extent in the way of instruments; he receives a reimbursement for his car expenses, which may be some little profit but not very much, so that his total gross income would be not more than £1,100, less out-of-pocket expenses and car expenses. Is that not what it comes to?

A.: Seven hundred pounds *plus* £50, that is £750, and car £200 to £250, would bring it up to £1,000 or £1,100 gross.

Q.: Out of that he has to pay his own car?

A.: Depreciation and cost of running his car.

Sir George Allard: Is not the point Dr. Mulvey was trying to make that the private practitioner not carrying on under the Government would require to receive fees from his patients to the extent of £1,200 to get the same return and pay for his *locum* and his car as a Government medical officer?

The Chairman: Yes, that may be the point.

Mr. Dovey: Apparently some parts of the country will not support a doctor on those terms, therefore the Government has provided one.

The examination of Dr. A. W. Shugg, which had not concluded at the adjournment on the previous day, was continued by Mr. Windeyer. Dr. Shugg described his experience as Government medical officer at Waddamanna.

Mr. Windeyer: Under the terms of your contract with the Government I think you were required there to provide services for all persons who were employed in connexion with the hydro-electric scheme?

A.: Yes.

Q.: What was your experience of the extent to which persons availed themselves of that service, and how did it compare with the way in which contract lodge patients availed themselves of their rights to services?

A.: I found that the work varied rather considerably. At Miena, where I had my headquarters, there were only about 150 men, who were practically no trouble at all. I suppose in the whole twelve months I was there I did not average more than one or two services for each individual. At the other two main camps the work was extremely heavy, and it was at those two sites that the majority of the men were situated. At Waddamanna I found the work was extremely heavy, and particularly they consumed colossal quantities of medicine—they seemed to thrive on it. They were a long way from hotels and apparently they seemed to like drinking medicine. There was very little alcohol in the medicines. The type of thing that happened was that either 6d. or 9d. was deducted from their wages each week, and a very large percentage of them adopted the attitude that they

were paying for this and they were going to get something out of it. In innumerable cases that was definitely done. At Miena that was not so; they seemed to be a better type of man there.

Q.: The places where they were most keen to avail themselves of the service were the more populous centres?

A.: Yes.

Dr. Shugg was then cross-examined by Mr. Dovey, mainly as to confidential matters and as to the accuracy of the count of heads made by the witness when he estimated that 536 of his patients would become insured persons under national health insurance.

Mr. Dovey: I suppose you always gave the same sort of service to your lodge patients as to your private patients?

A.: Not altogether.

Q.: Was it an inferior service?

A.: Of necessity, it had to be.

Q.: Of necessity, it had to be an inferior service?

A.: Because, as regards my private patients, I used to endeavour to have them after my lodge hours and make appointments with them. Supposing on Friday night there was a terrific rush of lodge patients from 7 to 9. Of necessity I could not give them the same time and attention as I could to the private patients.

Q.: What would be the most lodge patients that you would see between 7 and 9 at night, when you had the 1,100 or 1,200?

A.: I would sometimes see between 20 and 30.

Q.: In two hours?

A.: Yes.

Q.: You would not call that a terrific rush, would you?

A.: My word I would.

Q.: Including the repeat mixtures?

A.: Yes, because of those 20 or 30 there would necessarily be two or three who I considered looked as though they had something wrong with them, and I would have to spend perhaps a quarter of an hour or twenty minutes on one patient.

Q.: I suppose you got pretty good at spot diagnosis?

A.: One had to.

Q.: And you did?

A.: One had to.

Q.: And you did?

A.: Yes, and I may add that it is a very evil thing.

Q.: It is the universal practice?

A.: But it is an evil habit making spot diagnoses, definitely.

The Chairman: I suppose you will agree that a spot diagnosis by a medical man is better than no diagnosis by a medical man?

A.: Definitely.

Q.: And it becomes a question of giving the most that you can for the money which is spent. The ideal investigation, I suppose, would be something not less than half an hour to each patient, or something of that kind?

A.: Yes.

Q.: But would it be practical politics for a lodge to require an average of half an hour?

A.: Definitely not.

Q.: And the reason why it would not be practical politics is that the lodge would not have enough money to pay the doctor for it?

A.: Yes.

Q.: So we have to cut our medical cloth to suit our purse?

A.: Yes.

Mr. Dovey: I take it that your reluctance to recommence any form of contract practice is brought about by your reluctance to give to any patient a less efficient service than you are now giving to your private patients?

A.: That is one of the reasons, but not the only one.

Q.: What is the other one?

A.: Another reason I object to it very strenuously is on the question of night calls and what I call entirely unnecessary after-hour calls.

Q.: Did you find that that obtained in your lodge practice?

A.: To a certain extent, yes, and it does also, of course, in private practice, but in private practice you can say "No, I am not coming. You can either wait till the morning or get somebody else", particularly when you know the person, and what he is like, that he is an alarmist, or whatever it might be. Then you find a patient ill for a fortnight and ringing up at twelve o'clock at night. You definitely say that he can wait until the following morning, whereas, if you are bound under a contract with either national insurance or any other contract, you have to go.

Q.: I suppose that if a deterrent could be placed on a patient under national health calling doctors out at night, or calling them out unnecessarily, that would remove one of your difficulties?

A.: Largely.

Some argument between Mr. Windeyer and Mr. Dovey followed, the point at issue being the admissibility of evidence concerning the proceedings of a meeting of the Council of the Tasmanian Branch of the British Medical Association, at which Dr. Shugg did not attend but was represented by a proxy.

Mr. Windeyer: I suggest that my friend is not entitled to read out the whole minute, with the names of a lot of persons, and to take that as evidence that a lot of persons assented.

The Chairman: The only object he may have is to use it to refresh and fortify the witness's recollection of what he did authorize.

Sir George Allard: His recollection when he was not there?

The Chairman: I mean to fortify his recollection of what he authorized his proxy to do at the meeting at which that resolution was passed.

The Witness: I think I can answer it quite easily. The whole crux with regard to that resolution depended on this . . .

Mr. Dovey: I was not asking what the whole crux was. I was asking you did you authorize Dr. Whishaw to vote for the resolution?

A.: Yes.

Q.: And is this the resolution?

A.: I do not know what was ultimately put.

Q.: You were present at the next meeting and heard the minutes read and confirmed?

A.: Yes, I think so.

Q.: You appear in the minutes. This was resolved: "On the recommendation from the Branch council that this Branch considers that the capitation fee of 11s. proposed by the Commonwealth Government for medical services under the national health scheme is provisionally acceptable to the profession, but that the other modifications of the Act, including the 25% to country practitioners, suggested to the Government by the Federal Executive on the 21st May, are essential." The resolution was carried by 33 votes in favour and one opposed?

Dr. Mulvey: What was the date of that resolution?

Mr. Dovey. June 5. The resolution from the Branch, which was discussed at the meeting on May 30, was: "It was resolved unanimously that it shall be moved at the special meeting as the recommendation from the Branch council that this Branch considers that the capitation fee of 11s. proposed by the Commonwealth Government for medical services under the national health scheme is provisionally acceptable to the profession, but that the other modifications of the Act suggested to the Government by the Federal Executive on May 21 are desirable." That was the resolution of the council, and in the resolution passed by the Branch the word "desirable" was changed to the word "essential". The 25% increase was not mentioned. But the modifications suggested are to be found in that letter of May 21 from Dr. Hunter, the Federal secretary, which is already in. The special meeting on June 5 was called for that particular purpose. [To Witness]: So you thought that 11s. was all right?

A.: No, but I sent my proxy to agree to that provisional acceptance of the 11s. for one reason, which was this: The Federal Council had entered into a tentative agreement with the Commonwealth Government, and we in Tasmania were not conversant with 101 different details

of the whole scheme. So the attitude I adopted and the vast majority of the other members adopted was this . . .

Q.: Tell us your attitude; you cannot very well tell us the attitude of the others?

A.: My attitude, particularly being an ex-member of the Federal Council, was that we as a Branch should back up the Federal Council in all the efforts that it was making, and that we neither wanted to embarrass them nor to do anything else.

Q.: Nor to embarrass the Government?

A.: No, certainly not, provided that the Government entered into the matter in what we considered was a proper spirit. We did not want to embarrass anybody.

After further cross-examination by Mr. Dovey and reexamination by Mr. Windeyer the witness withdrew.

Dr. George Musgrave Parker, of Bellerive, a suburban and country district across the Derwent from Hobart, was examined by Mr. Windeyer.

Mr. Windeyer: You are a lodge medical officer to six lodges with nineteen branches?

A.: Yes.

Q.: I think your contract with them is purely oral, you have no written contract?

A.: That is so, I have no written contract at all.

Q.: What capitation fee do you get from the male members of the lodge?

A.: One pound per year.

Q.: That includes dependants?

A.: Yes.

Q.: Do you receive fifteen shillings for females?

A.: Yes, and if a widow, it includes her dependants.

Q.: Do you charge the lodge patients extras for excluded services?

A.: Yes.

Q.: Do you find you can in fact charge fees for excluded services and recover them from the patients?

A.: Yes, I charge fees for the excluded services.

Q.: Do you find you are able to do so successfully?

A.: Yes.

Q.: Do you charge full fees or modify them?

A.: I generally modify them for lodge patients.

Q.: You find that if you attempt to charge normal private practice fees to lodge patients for excluded services they go off to the public hospital?

A.: Yes, that is the usual thing. I really dare not charge full fees; if I do, I lose them.

Q.: The number of members on your lodge list is 105?

A.: Yes.

Q.: They have 189 dependants?

A.: Yes.

Dr. Parker gave further evidence of a confidential nature.

Mr. Windeyer: What do you say as to the services you have given to lodge members in comparison with those given to private patients, as to the quality of service?

A.: I endeavour to do my best to treat them just about the same.

Q.: Have you made a comparison of the value of the service which will be within the scope of the national health insurance scheme and that which is outside the scope?

A.: Yes.

Q.: From your experience, what do you say as to that?

A.: For each individual service I think the service without might be a little better than the service within the scheme, but taken in bulk I have far more services within than without the scope, and to compare the two is impossible. The services within the scope would be infinitely more valuable to me than the ones without.

Q.: You do very little of excluded services?

A.: Very little.

Q.: On your material you think that at per service, having regard to the time factor, the excluded service may be a little more valuable than the included service?

A.: I think it is probably a little more valuable than the included.

Dr. Parker estimated upon a count of heads that 122 out of 587 patients would become insured persons under national health insurance.

Mr. Windeyer: What do you think will happen to the dependants of national health insurance members; that is to say, persons who were previously under your contract practice, but who will not be insured under the national health insurance scheme?

A.: I think that a good many of the parents—probably one half—will keep up their lodge payments. From the conversations I have had with patients on the subject, I think that the balance, unless some provision is made under national health insurance for them, will drift into public hospitals. In any case, many of them will not be able to pay a decent fee.

Q.: Why do you say that they will not be able to pay for their services?

A.: I do not think that they will attempt to pay.

Q.: You have had conversations, I understand, with many of them?

A.: I have had conversations with some of the patients.

Q.: And in many cases you have found that they would have difficulty in paying?

A.: So they say. I can only take their word for it.

Q.: So that they will have difficulty in meeting their contributions to national health insurance and also making a friendly society contract?

A.: Yes.

Q.: You have heard the matter of mileage payment discussed from the point of view of a deterrent against unnecessary calls?

A.: Yes.

Q.: Do you say that a fee of 6d. per mile each way would be a sufficient deterrent?

A.: I do not think it is quite high enough.

Mr. Dovey: Not high enough to prevent the patient calling you?

A.: That is so.

Dr. Parker gave further evidence as to his experience of lodge practice and was then cross-examined by Mr. Dovey.

Dr. Francis James Bain Drake, of Huonville, was examined by Mr. Windeyer. Huonville is about 23 miles from Hobart and is an orcharding and saw-milling district on the Huon River.

Dr. Drake gave evidence as to his lodge practice, the capitation fee for which was 40s., of which the sum of 10s. was paid to a chemist for the provision of medicine. He stated that he had 36 lodge members, who had 90 dependants.

Mr. Windeyer: I think you find that lodge patients who will come under national health insurance generally go to hospitals at Hobart for services outside of the lodge agreement?

A.: Yes.

Q.: In fact, you facilitate their entrance by giving them a letter to the hospital authorities?

A.: Yes.

Q.: What do you think of the tendency of lodge patients to consult the doctor?

A.: They tend to consult the doctor for minor things which private patients naturally do not, as it is cheaper to get a bottle of medicine for a cold from the lodge doctor than to buy it from the chemist.

Q.: What arrangements have you in regard to mileage?

A.: I charge 5s. a mile one way over one mile.

Q.: That is day or night?

A.: Day and night.

Q.: That is per mile actually travelled?

A.: Yes.

Q.: The distance which you may have to travel in order to reach the patient may be very much greater in your area than the distance between your surgery and the patient, as the crow flies?

A.: Yes. There is a river running through the municipality with two bridges nine miles apart.

Q.: Do you get your mileage fee or do you find that people do not call you? What has been your experience of this mileage agreement in practice?

A.: If they cannot get to me because they are too ill they call me out, but most of them get a car from next door and come in to me.

Q.: So that the mileage fee is more of a deterrent?

A.: Yes, I have received very little in mileage fees.

Q.: What is your view of the amount necessary to charge to make the mileage fee an effective deterrent?

A.: It must at least be greater than the cost of the hire of a car.

Q.: Why do you suggest that as a basis of calculation?

A.: If it is cheaper to the patient to get me out ten or twelve miles—if, for instance, there were no mileage fees, it is much cheaper to get me out to see them than it is to hire a car to bring them in.

Dr. Drake gave further evidence, which was of a confidential nature. His estimate was that 37% of his private patients would become insured persons under national health insurance, but, making allowance for the consideration that, in theory at all events, these private patients would still be available to him for services outside the scope of service under national health insurance, the estimated loss of 37% was reduced to 32%.

Mr. Windeyer: And I think your opinion is that the effect of national health insurance will be to send the dependants of insured persons to public hospitals?

A.: Yes.

Q.: In your district the employed person is employed for the most part at a very small wage?

A.: He is employed most of the year, but not all the year, at 8s. a day, as an orchard hand.

Q.: And he gets his 8s. if the weather permits of his working?

A.: Yes.

Q.: And if the weather is bad and he is not able to work, he does not get any pay?

A.: No, and in certain slack times he is put off.

Q.: And from your knowledge of the conditions of the persons who are in employment in your district, you think, and I understand you have spoken to them, that there is no possibility of their being really available for private practice?

A.: No, none whatever.

Dr. Drake was then questioned at some length by the Chairman concerning the financial position and living conditions of these orchard hands.

Mr. Dovey: How many of these 8s.-a-day people are private patients of yours?

A.: A large proportion of my patients are in that region.

Q.: So that of the 643 people who attended as private patients, a large proportion were heads of households working as casual orchard employees at 8s. a day?

A.: Yes.

Q.: Do you think that you would be better off with some form of contract service with them?

A.: In some ways.

Q.: In respect of each and every one of them, if you got a certain half a guinea, whether they came to you or not, would not you be better off than you are now, with that class of person?

A.: Yes, but I cannot say definitely what proportion those 8s.-per-day people are in the total amount.

Q.: Quite so, but as far as the 8s.-per-day casual workers are concerned, you treat them as private patients and give this love of God service?

A.: I find that the great majority of them pay me.

Q.: But you would be better off with one consultation fee of 10s. 6d. per person *per annum*, whether he saw you or not, than you are at present, as far as they are concerned?

A.: As far as they are concerned, yes.

Dr. Drake was further cross-examined by Mr. Dovey and then withdrew.

Dr. James Scott Reid, of Hobart, was examined by Mr. Windeyer regarding his lodge practice. He estimated the proportion of lodge members to dependants on his lists as 1 to 1·3.

Mr. Windeyer: Is there any income limit?

A.: I believe there is, but I have never enforced it.

Q.: You know that some of your lodge patients are in very good circumstances?

A.: Yes.

Q.: There is no night fee in your lodge agreement?

A.: No.

Q.: I understand there is a right to ask for a conveyance at night?

A.: Yes.

Q.: It seems to be a relic of the past, when the doctor could not be expected to get out his own horses after the coachman had gone?

A.: Yes.

Q.: That is the deterrent against unnecessary night calls, and you are entitled to ask a patient to provide you with a conveyance instead of you getting out your own car?

A.: Yes.

Q.: Occasionally, for the purpose of a deterrent, you do in fact ask that?

A.: Yes.

Q.: In reference to the question of whether lodge patients tend to go to public hospitals for treatment and extras, or come to a doctor, I understand your view, from your experience, is that the men readily go, and tend to go to public hospitals, but the women prefer whenever they can to come to a doctor as private patients?

A.: Yes, it is more especially so in operations. The women do not like going to a public hospital ward, but prefer to go to a private hospital. Even though there may be a ward there, it has only three or four beds in it.

Q.: You think it is partly for reasons of privacy, and also you have found that women dislike a change of doctor?

A.: Yes.

Q.: You are speaking of the tendency of lodge patients to make use of hospitals as in-patients in respect of excluded services?

A.: Yes, as in-patients.

Q.: Do you find your lodge patients prone to visit you unnecessarily?

A.: Some of them are very prone. I have seen one or two of them fifty-two times a year for ten years—once a week—but the majority of them are very reasonable.

Q.: Do you find that a large amount of your lodge work is really giving orders for cough mixtures and indigestion mixtures, and so on?

A.: Yes, they feel they have to get the medicine. If they have a cough and they are not off their food and are not ill, and they have to get a cough medicine, they know they have to get an order from me before they can get medicines from the dispensary, and they come to me to sign an order.

Q.: They come to you not so much as a doctor, but as a person who can sign an order on a dispensary?

A.: Yes.

Q.: Do you enforce the mileage clause or provision of your lodge agreement?

A.: Yes.

Q.: What is your mileage charge?

A.: The mileage chargeable is 5s. a mile in the day-time and 7s. 6d. at night outside a three-mile limit from the post office one way.

Q.: Do you find that is an effective deterrent against unnecessary calls?

A.: Yes, I have had very few calls to my lodge patients outside the three-mile limit.

Dr. Reid then gave evidence of a confidential nature, and his examination had not concluded when, at 4.35 p.m., the Commission adjourned until Wednesday, November 16, at 10 a.m.

Wednesday, November 16, 1938.

Dr. Reid was further cross-examined by Mr. Windeyer.

Mr. Windeyer: You told us yesterday about the mileage rate in your contract practice, and you told us that you considered it was an adequate tariff?

A.: Yes, I think so.

Q.: Do you think that 6d. per mile each way, commencing after the third mile, would be a sufficient deterrent?

A.: It would not in my practice, because the majority of calls that I get outside the three-mile limit are between three and four miles; therefore, I would only get 1s. for driving eight miles.

Q.: I suppose that if a person lived a considerable distance off, 6d. per mile would have a greater deterring effect at all events?

A.: Yes, but it would barely pay car expenses. It would not pay anything for travelling time.

Q.: I was considering the matter from the point of view of its deterrent value. Now I understand you estimate that you work 56 hours a week for 50 weeks in the year?

A.: Yes.

Dr. Reid was cross-examined by Mr. Dovey and then withdrew.

Dr. William Albert Fleming, of Burnie, was examined by Mr. Windeyer.

Dr. Fleming gave evidence of a confidential nature as to his lodge and private practices. He said that the capitation fee for lodge members was 20s. for males, 15s. for single females and 20s. for females with dependants.

Mr. Windeyer: Do you receive payment for extras outside the lodge agreement?

A.: Yes.

Q.: What is the position of your practice in regard to that; do you look for that work?

A.: No, I am afraid we do not. We do not entice extras, actually.

Q.: You do not look specially for them?

A.: No, we do not look for extras.

Q.: When you do them, do you get the ordinary private fee?

A.: I should say generally, yes, we do. It may be a little lower, but not very much lower.

Q.: What happens, as a rule, to lodge patients who want extras?

A.: Those who come along asking for extras, we give them the services, but we do not entice them to accept any extras. In other words, we allow them to go to public hospitals if they wish to.

Q.: And where do they go, in fact?

A.: There are two public hospitals in our vicinity.

Q.: One is the Spencer Hospital, which is at Wynyard, 12 miles from Burnie, and the other is the Devon Hospital, at Latrobe?

A.: Yes, about 40 miles away.

Q.: But you find that, in fact, a number of your lodge patients do go to Latrobe?

A.: Yes. That is, excluding workmen's compensation cases. We do definitely hold on to those.

Q.: But other extras go to one of the hospitals?

A.: Generally.

Q.: I understand you do not consider that the lodge capitation fee under your present agreement is adequate?

A.: We do not.

Q.: And in fact you approached the lodge about 12 months ago for an increased rate?

A.: Perhaps a little longer.

Q.: But the lodge was not prepared to pay anything more?

A.: That is correct.

Q.: And for various reasons you did not think it was politic to force the issue at that time?

A.: That is correct.

Further confidential evidence was given by Dr. Fleming, who had prepared a great deal of statistical evidence from his records. Of 825 private patients, he considered 237 would become insured persons under national health insurance. On taking into consideration the excluded services under national health insurance he estimated that his loss of private practice would be 25%.

Mr. Windeyer: That is, as nearly as you can estimate it, the diminution in your private practice which will result from national health insurance, on the assumption that the insured persons will be still available and come to you for their excluded services?

A.: Yes.

Q.: Do you think in practice they will come to you for their excluded services?

A.: I should say to the same extent as lodge patients do.

Q.: Have you given any attention to what will be the position of the dependants of the persons who come under national health insurance?

A.: I have questioned several of my members and they all expect that some scheme has to be formed for their dependants.

Dr. Fleming then gave details of an arrangement that had been recently entered into with the Associated Paper and Pulp Company, Limited, which had recently commenced operations at Burnie.

Dr. Fleming was then cross-examined by Mr. Dovey.

Mr. Dovey: You say that lodge patients do not receive as good service as private patients; why is that?

The Chairman: There is no need to expatiate upon that. One has to exercise a little common sense about the matter, and I shall apply what I call my common sense to it. Of course, they do not get the same amount of attention as private patients do. I have heard it said by a lot of medical practitioners before this tribunal that they give as much time to lodge patients, on the average, as they do to private patients.

Mr. Dovey: They have all said it on the mainland, with one single exception.

The Chairman: They have said that they have done it because they feel that they can pay another visit, and so on, without running the risk of nasty imputations. In practice, I am satisfied—I do not care how many doctors say the contrary—that, on the average, the lodge patient does not get as much time from the medical man as the private patient does, and it would be unreasonable for him to expect it.

Mr. Dovey: I think so, if I may say so with respect.

The Chairman: There are some matters about which we do not require a lot of evidence.

Mr. Dovey: This is the first time that there has been any pronouncement from any member of the Commission.

The Chairman: I am only speaking for myself.

Mr. Dovey: In respect of this matter, the evidence has been almost all one way, and this is not the only matter on which the evidence has been all one way. I will not say any more at the moment.

The Chairman: This gentleman has been quite frank about the matter.

Dr. Mulvey: I concur entirely in what the judge says in this regard. But there is this position to be considered, that a medical practitioner does give a family service to a lodge patient and his dependants which he does not give to a private patient. That is, he pays more visits, and gives them attention by what would be considered unnecessary calls by a private patient. On the other hand, the amount of time given to a patient who comes for a consultation or whom he visits would not be the same as for a private patient, as he is already familiar with the family history of the patient.

Mr. Dovey: I have now heard what two-thirds of the members of the Commission have to say, but, as I have said, I do not propose to labour the matter any further.

Sir George Allard: I think that the doctor's answer was quite satisfactory, and that there was no injustice done to the society members.

Mr. Dovey: I am not concerned with whether there has been any injustice done, if I may say so.

Sir George Allard: It would be an injustice if they did not get proper treatment. He said they had good treatment, and that is quite sufficient. They get good service.

Mr. Dovey: All I had in mind was that, until we came to this State, with one single exception, over one hundred doctors being called, the doctors said that the lodge patients got as good, if not better, service than the others. I have finished with the witness.

Dr. Fleming was further examined by Mr. Windeyer and was then cross-examined by Mr. Bowie Wilson on statistical matters with reference to the witness's records.

Dr. Jack Rail Robertson, of Deloraine, was examined by Mr. Evans.

Deloraine was described as a rural town with a population of about 2,000, surrounded by a completely rural area, about 35 miles from Launceston.

Dr. Robertson gave confidential evidence concerning his lodge and private practices.

Mr. Evans: What is your experience of the tendency of lodge patients to go to hospitals for extras?

A.: For major work only. If it is minor extra work, they prefer to remain in the town, if possible.

Q.: I think you find that you are frequently consulted by lodge patients for trivial complaints?

A.: Yes.

Q.: You also do not think that the capitation fee is adequate for the services rendered, but you feel that you get counterbalancing benefits. What benefits would they be?

A.: I find that a satisfied lodge patient is probably one of the best scouts for my private work. He brings a lot of private work to you, and also you have the regular quarterly cheque coming in to balance the budget. Further, there is a certain amount of concessional work for the lower classes in the district.

Q.: Coming now to the mileage clause in the lodge agreement, I understand that the limit is three miles and that the rate is 5s. per mile up to eight miles, and 2s. 6d. per mile above that, one way in each case?

A.: Yes.

Q.: Do you find that the imposition of that mileage rate has any deterring effect?

A.: Very definitely so.

Q.: While we are on the mileage, for private patients, what is your rate?

A.: Five shillings per mile after the first mile.

Q.: Also on mileage, what do you say to this suggested mileage fee to be paid by the patient—6d. per mile each way outside the three-mile limit?

A.: Personally I do not think it is much of a deterrent. A patient living five miles out would have to pay me only 2s. I do not think it would deter him very much.

Q.: What would you suggest as a minimum?

A.: I should say something in the region of 2s. 6d. one way might help to stop them a little.

Dr. Robertson estimated the loss of private practice he expected upon the introduction of national health insurance in a similar manner to the other witnesses and arrived at the figure of 31%.

Mr. Evans: Taking the dependants and the members who come under national health insurance, what do you think they will be able to pay you as private patients, or do you think you will retain them as private patients?

A.: I do not think so.

Q.: Where do you think they will go?

A.: They will tend to go to hospitals for most of the treatment.

Mr. Dovey: Which hospital?

A.: Launceston or the Devon Hospital.

Q.: As in-patients or out-patients?

A.: As out-patients.

Mr. Evans: I think you stated earlier that you found a large number of trivial services were asked for by your present lodge patients. What would you say as to the same tendencies under national health insurance?

A.: I think it will be considerably aggravated and they will be chasing us for even more trivial complaints.

Q.: Do you think that any substantial proportion of your lodge patients will be able to continue in membership of their lodges, or their dependants, after the national health insurance scheme comes in?

A.: Quite a number of them will not be able because they will not be able to pay both contributions.

Q.: What do you think their dependants will do?

A.: They will either become a non-paying class entirely or they will have to utilize the public hospitals as much as possible.

Dr. Robertson was cross-examined by Mr. Dovey upon the figures he had given and the estimates which he had made.

Dr. Wilfred Wanostrocht Giblin, of Hobart, was examined by Mr. Windeyer. He gave confidential evidence concerning his practice and also gave evidence concerning certain attempts that had been made to secure from the friendly societies an increase in the metropolitan capitation rate of 20s. for males and 15s. for females.

The Commission adjourned at 12.15 p.m. to the Commonwealth Offices, Melbourne, on Monday, November 21, 1938, at 10.30 a.m.

Correspondence.

INFANT WELFARE.

SIR: In a speech advocating the formation of an infant welfare centre a female doctor (childless) stated that maternal instinct was not sufficient guide in bringing up children; that it was better for young mothers to get advice from nurses trained in welfare work than from "granny"; that before the centres commenced their work the infant mortality was 10% and had been reduced to 3·6%; and that in view of the falling birth rate it was important to extend the influence of the centres so that a higher proportion might be saved. This stock speech has been used thousands of times by hundreds of people, and I have never yet known anyone rise up and call it piffle.

A less misleading statement would be that the maternal instinct should be carefully fostered, since children cannot be successfully reared without it; that "granny's" advice is probably better worth taking than that of any childless woman, however highly trained; and that since welfare clinics began the number of births has fallen off to such an extent that the population is bound to decrease unless assisted by immigration. The whole trouble is that of those given official authority to teach mothers how to rear their children, probably not one in five hundred has ever suckled a baby. They have worked out a system which seems to suit babies very well, but the viewpoint of a mother is beyond any male or childless female. With the best intentions and the most enlightened views they are capable of forming, they have devised a system which so little satisfies the maternal instinct and gives so little pleasure to the mothers that the result is better babies and fewer. If the Australian bride prefers bridge, golf or dancing to baby rearing it is not because she is giddy or frightened or lazy, but because she gets more fun that way.

It is impossible for me, a male, to say what the missing ingredient is; but I think I can suggest a way to supply it. Let all infant welfare movements be staffed with trained grandmothers. Suitable women for training are not lacking. The training would no doubt have to be altered to suit their stubborn prejudices, and after that it should be really valuable.

Yours, etc.,

J. B. HOGG, M.B.

Koorda,
Western Australia,
November 11, 1938.

A CONSIDERATION OF GENERAL ANÆSTHESIA FOR DENTAL SURGERY.

SIR: In reply to Dr. Watkins's letter in the journal of November 12, 1938, one must remark that common sense is not based on fashion. Endotracheal insufflation anæsthesia has been superseded by the more efficient, though slightly more difficult, endotracheal inhalation technique, with complete isolation of the respiratory tract from the oral and nasal cavities. No reliance is placed on the back-flow of air to prevent the entry of foreign matter into the bronchial tree. It cannot get there. Does blood never pass down the trachea with efficient endotracheal insufflation anæsthesia, with continuous outflow of air? One cannot answer with Gilbert's "Well, hardly

ever!" It occurs frequently in the hands of anæsthetists as careful as myself and possibly as careful as Dr. Watkins, but never since adopting the inhalation technique. Has Dr. Watkins never had to wash blood clot from the eye of a catheter, clot so firmly adherent that its presence cannot be explained as clot picked up in the mouth during removal of the catheter? Incidentally, there should be no clot in the pharynx at this stage.

How often does bronchial secretion collect in sufficient quantity to cause trouble? Surely very seldom! I have met this trouble only in patients with bronchiectasis. One has at hand a suction apparatus and it is a simple matter to remove any secretion. There is in fact a catheter connexion in which provision is made for suction of the bronchial tree without interrupting the anæsthesia; I have had one in my bag for years and have used it twice.

I would invite anyone to compare the condition at the end of operation of two patients anæsthetized by these two methods.

Respiration in the inhalation technique has been quiet throughout and at atmospheric pressure, requiring the expenditure of the minimum of energy by the patient. Respiration in the insufflation technique has been forceful throughout, expiration taking place through a narrowed glottis and against the pressure of the delivery of the anæsthetic mixture—up to twenty millimetres of mercury. The patient has expended much energy thereby, as is demonstrated by his sweat-saturated garments and comparative exhaustion.

Endotracheal insufflation anæsthesia confers relative safety only, against the absolute safety of endotracheal inhalation anæsthesia.

Yours, etc.,

DOUGLAS G. RENTON, M.B., B.S.

12, Collins Street,
Melbourne,
November 14, 1938.

THE FIRST FIFTY-TWO NAMES ON THE MEDICAL REGISTER OF SOUTH AUSTRALIA.

SIR: On page 737 of your issue of October 29, 1938, mention is made of my grandfather, Dr. William Horatio Sholl, who died in my infancy. The name is annoyingly misspelt Scholl, which it never was. My uncle, Lionel H. Sholl, is also mentioned over the page as Scholl. Our name goes back in family records in Cornwall to the fourteenth century, previous records having been destroyed by a Spanish raiding party, which burnt a church. The "Scholl" habit, as I have pointed out to Professor Cleland, has been created through the German American who calls himself "Dr. Scholl" and advertises "Footease". My relation to Dr. W. H. Sholl is grandson; my late father, Richard, was his second son.

Yours, etc.,

R. F. SHOLL.

409, St. Kilda Street,
Elwood, S.3,
Victoria.
November 13, 1938.

THE ECONOMIC ASPECT OF INDUSTRIAL SURGERY.

SIR: Dr. Bell Allen, in his article on "The Economic Aspect of Industrial Surgery", published in the journal of November 12, 1938, has opened a subject which is apparently not sufficiently borne in mind by many members of the profession. Congratulations are due to Dr. Bell Allen, therefore, for bringing this matter so ably before us. There are, however, certain points in which many surgeons must differ from him. He states that in dealing with injuries to the hands, if all foreign materials can be removed by excision, healing by first intention will result in 100% of cases. While admitting that his advice here is the very best, the feeling remains that to get primary healing in every case is expecting a great deal. In many cases, if a thorough excision is done, considerable areas must be left without any covering of skin, and in these immediate healing cannot result. Most surgeons will agree, I think, that in one hundred consecutive clean operation cases primary healing is not likely in every case. My experience has been that in extreme injury to the hand or any other part of the body appropriate splinting of the part is of very considerable value in assisting rapid healing.

Dr. Allen is anxious to learn the reason for keeping the patient in bed for more than a few days after removal of a medial meniscus. Personally I have found that if a patient is allowed out of bed after three or four days the knee joint becomes distended by synovial fluid and the patient feels uncomfortable and loses confidence. As the wound is continually being stretched, and as the knee joint is distended, many cases develop a leak of synovial fluid or continually exude a little serum, so that healing is delayed for three or four weeks. If on the other hand the patient remains in bed for two weeks, actively moving his knee joint after the fifth or sixth day, or more particularly making active contractions of his *quadriceps femoris* muscle, he is able to get out of bed on the fourteenth day with a firmly healed wound, no excessive fluid, and at least 90° of active movement. He is able to walk painlessly without any support, and in two or three days after leaving his bed is able to work quite naturally. The period of disability is usually not more than six or seven weeks for labourers, and clerical workers often resume work three weeks after operation.

In the case of Pott's fracture it has been proved conclusively in the fracture clinics at the Royal Prince Alfred Hospital, at Marrickville Hospital and at Parramatta Hospital, that, of the present methods of treatment, that which shortens the time of disability to the greatest extent is the application of an unpadded plaster of Paris splint in which an iron heel or other walking appliance is fitted. Treated in this manner, atrophy of muscles and joint stiffness occur only to a slight extent, whereas treatment with a Delbet plaster largely defeats its own purpose by allowing the foot to become œdematous. Immobilization of the ankle joint does not cause nearly the same degree of joint stiffness as do continued swellings due to œdema. In addition, it was found that the Delbet plaster does not sufficiently fix the fracture site in cases in which the posterior portion of the articular surface of the tibia has been fractured.

Dr. Bell Allen suggests that statistics be published to indicate the period of incapacity following various injuries. It has been found that in the case of fractures the period is so variable, and depends so much on various complications, that a very large series of cases would be necessary to determine a reasonable average. It has been found, for instance, in our fracture clinic at Marrickville Hospital that in cases of Colles's fracture the period of disablement from manual labour varied from four weeks to forty-seven weeks, and that the average in 143 consecutive cases amounted to six weeks.

Yours, etc.,

W. MCCLEMENTS CALLOW.

135, Macquarie Street,
Sydney,
November 14, 1938.

ELECTION OF THE QUEENSLAND BRANCH COUNCIL.

SIR: I wish to protest strongly against the method of conducting the ballot for the forthcoming election of the Queensland Branch Council. Without casting any insinuations I would point out that all ballot papers bear a number and are therefore not necessarily secret. It is possible for every member of the council to know the names of those who voted against him. The ballot, like Cæsar's wife, should be above suspicion.

I have always understood that the British Medical Association is a democratic institution, of which a secret ballot is a necessary principle. Evidently in British Medical Association circles we are suffering from the prevailing perversion of democracy to bureaucracy.

Yours, etc.,

T. M. S. HALL.

Taylor's Buildings,
Ruthven Street,
Toowoomba,
Queensland.
November 15, 1938.

SIR: In reply to the complaint made in the letter of Dr. T. M. S. Hall under date 15th instant, I have to state that the ballot is absolutely secret. No record of the number on any ballot paper sent out to members is kept or noted in any way. The system of numbering of ballot papers has always been done and is merely used to check the total number sent out.

The council is not interested in the personal vote of any member.

Yours, etc.,

HORACE W. JOHNSON,
Honorary Secretary.

British Medical Association House,
225, Wickham Terrace,
Brisbane.
November 21, 1938.

Obituary.

TOM FARRANRIDGE.

TOM FARRANRIDGE, who died on August 22, 1938, was a graduate of the University of Sydney. He had a brilliant academic career, winning first-class honours and the University Medal at his graduation in 1917. He served as a resident medical officer at the Royal Prince Alfred Hospital from 1917 to 1918, then left for service in the Great War with the rank of captain in the Australian Army Medical Corps. He returned to Australia in 1919 and was appointed senior resident medical officer at the Royal Hospital for Women, a position that he held until 1922, when he was appointed honorary assistant surgeon to that institution. In the following year he was appointed honorary assistant surgeon to the Royal Prince Alfred Hospital, and tutor in surgery in the University of Sydney. It is probably for his teaching that he will best be remembered. He had the rare gift of the ability to impart learning; in addition, he was cheerful and entertaining; for these reasons he was always sure of a large audience of students on his visiting days at the Royal Prince Alfred Hospital. He became tutor in clinical surgery in 1931.

In addition to being a Bachelor of Medicine and Master of Surgery of the University of Sydney, he was a Fellow of the Royal Australasian College of Surgeons and a Member of the College of Obstetricians and Gynæcologists of London.

He was honorary visiting surgeon to the New South Wales Masonic Hospital and honorary consulting surgeon to the Hornsby and District Hospital and the Ryde District Soldiers' Memorial Hospital.

He took a great interest in surf life-saving and was a member of the Bondi Surf Life Saving Club. He was its honorary medical officer for many years.

After leaving his position of resident medical officer at the Royal Hospital for Women, he practised as a surgeon in Macquarie Street, Sydney, continuing in active work until a week or so before his death. He had a wide knowledge of surgery and was a dexterous operator.

During the last few months of his life Farranridge won the highest admiration from his colleagues for the manner in which he carried on in spite of the most distressing physical disabilities. His continued cheerfulness and courage in adversity were an inspiring example to those with whom he came in contact.

He was unmarried.

Dr. Alan B. Lilley writes:

Dr. Tom Farranridge served on the surgical staff of the Royal Prince Alfred Hospital from 1923. At the time of his death he was the senior of the honorary assistant surgeons on the staff of the hospital.

He was a most charming gentleman, always to be relied on to carry out his duties with the utmost efficiency. He had a particular appeal to the medical student, as he interested himself in the teaching side of his hospital duties.

We all remember his great capacity as a tutor in surgery, and many of us owe our success in surgical examinations to the painstaking efforts of Dr. Farranridge in assuring us of a good knowledge of this subject.

One could not but admire the outstanding courage with which our late colleague continued to carry out his work while suffering the greatest physical strain.

His passing, which we deeply mourn, is a severe loss to the Royal Prince Alfred Hospital; and he has left behind a tradition of loyal service to the indigent sick. His life was rich in accomplishment, and he built for himself a lasting place in the memories and esteem of his colleagues.

Dr. R. S. Irwin writes:

It was with genuine regret that I learned of the death of Dr. T. Farranridge. As one closely associated with him for many years, both as a student and later as his house surgeon at the Royal Prince Alfred Hospital and at the Royal Hospital for Women, I should like to add some token of appreciation to those you will undoubtedly receive from his contemporaries.

As surgical tutor at the Royal Prince Alfred Hospital he took charge of those students who were just commencing their hospital work. His classes were always informal and his students always happy, and he soon dispelled the natural nervousness that most students feel when taking on that new type of work.

His out-patient afternoon at the Royal Prince Alfred Hospital was always overcrowded with students, many of whom remained behind after the last patient had gone, to take afternoon tea with him, smoke his cigarettes and listen to his discussions on any subject which might be brought up. This speaks volumes for his popularity as a man and as a teacher. He never seemed happier than when surrounded by students or when mixing with the resident medical officers in their quarters, and it was never any trouble to him to explain any point in surgery that was not clear to any of us. With his never-failing smile and irresistible good humour he seemed to have captured the spirit of perpetual youth, and it is all the more of a shock to find that one who was so well liked has departed this life at such a comparatively early age.

I have no hesitation in saying that there are many young doctors in Australia who will cherish his memory and be ever grateful to him for his help and advice, which were always given so generously.

LESLIE SWINNERTON DUKE.

WE regret to announce the death of Dr. Leslie Swinnerton Duke, which occurred on November 9, 1938, at Mendooran, New South Wales.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1925 to 1935*, of Queensland, as duly qualified medical practitioners:

Arden, Felix Wilfrid, M.B., B.S., 1931, M.D., 1934 (Univ. Adelaide), M.R.C.P. (London), 1936, Brisbane.

Patterson, Hamilton Stuart, M.B., Ch.B., 1938 (Edinburgh), L.R.C.P., L.R.C.S., 1938 (Edinburgh), L.R.F.P.S., 1938 (Glasgow), Ipswich.

Powell, Keith William, M.R.C.S., 1938 (England), L.R.C.P., 1938 (London), Brisbane.

Corrigenda.

NASAL SINUSITIS IN CHILDREN.

DR. W. KENT HUGHES points out an error in his letter on nasal sinusitis in children, which appeared in the issue of November 12, 1938. The first sentence should read: "Over thirty years ago I gave up lavage except in cases of acute sinusitis". In the letter, as published, the word "acute" was missing.

REPORT OF A MEETING OF THE BRITISH MEDICAL ASSOCIATION AT WANGARATTA.

DR. G. A. PENINGTON has drawn our attention to an error in the report of his remarks made at a meeting of the Victorian Branch of the British Medical Association at Wangaratta on July 16, 1938. The error appears at page 836 of the issue of November 12, 1938. Dr. Penington is reported as saying that "2·5 grammes of histamine should be injected". This should read: "0·25 milligramme of histamine should be injected".

Diary for the Month.

DEC. 1.—Western Australian Branch, B.M.A.: Council.
DEC. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
DEC. 6.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 7.—Victorian Branch, B.M.A.: Annual Meeting.
DEC. 7.—South Australian Branch, B.M.A.: Council.
DEC. 8.—New South Wales Branch, B.M.A.: Branch.
DEC. 9.—Queensland Branch, B.M.A.: Annual Meeting.
DEC. 13.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 14.—Victorian Branch, B.M.A.: Council.
DEC. 16.—Queensland Branch, B.M.A.: Council.
DEC. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii to xx.

ANTI-CANCER COUNCIL OF VICTORIA: Executive Medical Officer.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

COMMONWEALTH OF AUSTRALIA, DEPARTMENT OF THE TREASURY, CANBERRA, FEDERAL CAPITAL TERRITORY: Chief Medical Officer.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.

QUEEN VICTORIA MEMORIAL HOSPITAL, MELBOURNE, VICTORIA: Resident Medical Officers.

REPATRIATION COMMISSION: Resident Medical Officer.

ROCKHAMPTON HOSPITALS BOARD, ROCKHAMPTON, QUEENSLAND: Resident Medical Officer.

ROYAL HOBART HOSPITAL, HOBART, TASMANIA: Resident Medical Officer.

ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

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